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OF
MEDICAL JURISPRUDENCE

TENTH EDITION

EDITED BY

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J. & A. CHURCHILL LTD.
104 GLOUCESTER PLACE, W.1.
1948

1st	Edition	1865	Prof. A. Swaine Taylor.
2nd	,,	1873	do.
3rd	,,	1883	Revised by Dr. T. Stevenson.
4th	,,	1894	do.
5th	,,	1905	Revised by Dr. F. J. Smith.
6th	,,	1910	do.
7th	,,	1920	do.
8th	,,	1928	Revised by Prof. Sydney Smith and Dr. W. G. H. Cook.
9th	,,	1934	do.
10th	,,	1948	do.

1375

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N48.2

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PREFACE TO THE TENTH EDITION

In this, the tenth edition of Taylor's *Medical Jurisprudence*, recent advances in the various medical and ancillary sciences, together with recent legislation by the British Parliament, have necessitated a considerable amount of alteration and revision in every part of the work. Whereas the essential features of "Taylor" have been maintained, the opportunity has been taken to revise and to modernise the text, and to clarify a number of passages which were previously somewhat obscure. In some parts, the arrangement of the book has been altered; certain sections have been entirely re-written; and a great deal of new material has been included. We have not considered it desirable to delete references to old cases simply because they are old; but where these could, with advantage, be replaced or supplemented by more recent case histories, this has been done. It is hoped that these changes will prove to have enhanced the value of the work.

In response to the suggestions of a friendly reviewer of the previous edition, we have omitted a large proportion of the technical details formerly given in the chapters on Insurance and Workmen's Compensation. The saving of space thus effected has been utilised with advantage in other parts of the work.

A further consideration which justified a curtailment of the space formerly devoted to the Workmen's Compensation Acts is the impending supercession of that Act by the new National Insurance (Industrial Injuries) Act, 1946.

The new Act will bring about a much needed change in the methods of dealing with compensation for injuries to employed persons. The superseded legislation, although of considerable social importance, tended to affect adversely many claimants of compensation. The examination by medical men for the two opposing parties; the giving of evidence in Court, and the long-drawn-out nature of the enquiry tended to produce a neurosis or to accelerate an existing tendency to a neurotic state which was not desirable. The conflicting opinions which medical referees and judicial authorities had to consider were neither creditable to the medical profession nor helpful to the ends of justice. As emphasised throughout the present work, any suggestion of the existence of partiality in a medical witness would be entirely contrary to the ethics of the profession. The new Act has been dealt with very briefly in its medico-legal applications, which is all that is possible or necessary at the present stage; it would seem, however, that many of the defects of the former legislation have been remedied.

No attempt has been made to provide detailed information regarding certain other legislative enactments, including the National Health Service Acts, which are important from both the medical and legal points of view. It does not appear, however, that they create any distinctly medico-legal problems or affect principles which are not already dealt with; and for that reason it does not seem necessary to consider them at length, at the present stage, in a work on medical jurisprudence.

The following statutes, among other, which affect in varying degrees practitioners of medicine and surgery have been noticed in completing

the revision of the text : The Public Health Act, 1936, the Matrimonial Causes Act, 1937, the Factories Act, 1937, the Food and Drugs Act, 1938, the Mental Deficiency Act, 1938, the Children and Young Persons Act, 1938, the Infanticide Act, 1938, the Road Traffic Act, 1938, the Cancer Act, 1939, the Pharmacy and Medicines Act, 1941, and the Nurses Act, 1943.

The recent decisions of the English Courts of Law which have been referred to in the new edition include the decisions of the House of Lords in *General Medical Council* v. *Spackman*, and in *Lindsey C.C.* v. *Marshall*, the decision of the Court of Criminal Appeal in *Rex* v. *Bourne*, and the decisions of the Court of Appeal or of the High Court of Justice in *Mahon* v. *Osborne*, *Gold* v. *Essex County Council* and *Morris* v. *Winsbury-White*.

In the toxicology section, we have taken the opportunity of dealing with some of the drugs which, introduced into medicine in recent years, have been the causes of toxic reactions or fatalities, of which the sulphonamide drugs form the outstanding example. The general description of procedures in toxicological analysis has been re-written with a view to giving a clearer account of the available methods and their application. Throughout, newer methods of analysis have been described, but older methods have been deleted only where they are known to be unreliable. Thus the Marsh-Berzelius and Gutzeit tests for arsenic have been retained, but have been supplemented by a description of the newer titrimetric method.

The accounts of the mode of action and symptomatology of the various poisons have been carefully revised so as to include all important facts, but to eliminate the repetition that was to be found in earlier editions. Illustrative case records remain a feature of this section ; and have been chosen because of completeness or because of their striking nature rather than on the basis of date. Often old cases provide a fuller description than modern ones and when this is so, they have been retained.

The statistics given in this edition have been culled from the latest official returns published in respect of England and Wales.

We acknowledge with gratitude the invaluable help in the sections relating to Coroners and witnesses' fees which we have received from Mr. W. Bentley Purchase, M.C., and Master W. Valentine Ball, O.B.E., respectively. We have to thank Dr. Robert Richards for his help in the chapter on alcoholic intoxication, Dr. James Davidson, for his help in the chapter on blood grouping, Dr. F. S. Fiddes for his general help throughout, and many correspondents for their helpful suggestions.

SYDNEY SMITH.
W. G. H. COOK.

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THE PRINCIPLES AND PRACTICE

OF

MEDICAL JURISPRUDENCE

PART I

SEXUAL MEDICAL JURISPRUDENCE. OBSTETRIC AND CRIMINAL

Sex as a fact in identity, and the means we have for determining the sex of a person, when this is open to doubt, have already been fully discussed in Vol. I, pp. 110 and 151. We have now to discuss the concrete relationships of the two sexes :—the woman as a possible wife or mother, the man as a husband and possible father, the union and the dissolution of the union of husband and wife, unlawful union of man and woman, or of man with beast, the results of the union of man and woman and the methods by which these results may develop and reach the outer world.

Certain sexual problems of a general nature, the solutions of which have a bearing on several special cases, are discussed first and are followed by a review of the subjects of special interest.

The general questions are :—Is this man or woman capable of intercourse ? If so, can such intercourse be fruitful ? Has this woman had sexual intercourse ? Is this woman pregnant ? Has this woman had a child either recently or at some previous time ?

We shall therefore discuss our medico-legal problems in the following order :—

Impotence and Sterility.
Virginity and Defloration.
Pregnancy and its Duration.
Parturition or Delivery.
Dissolution of Marriage.
Legitimacy and Paternity.
Rape.
Abortion.
Natural Birth.
Infanticide.
Unnatural Offences.

CHAPTER I

IMPOTENCE AND STERILITY

Impotence

Impotence may be rigidly defined as "the incapability of either sex to allow or grant to the other the legitimate gratification of the sexual desire." The difference between this and sterility must be carefully borne in mind (*vide infra*), for the one may be a bar to union or a cause for divorce, the other is neither one nor the other in the eyes of the law.

The causes of impotency may be thus classified :—

General or functional, unconnected directly with the sexual organs.	Age Illness. Emotion.	Any of which may lead to, or cause, temporary, or permanent, incapacity.
Local or organic, in direct connection with the sexual organs.	Congenital or acquired conditions.	Curable. Doubtfully curable. Incurable.

All of which groups must be noticed separately.

Age as a cause of Impotency. *In Females.* As the woman is a passive agent in the sexual act, there can be no limit to the oldest age at which she can be potent to allow the act, provided she be free from disease or deformity ; whether she has her sexual instincts still preserved or not has nothing to do with the matter, though we have no reason to believe that mere age ever extinguishes them while life remains. As regards the youngest age, before sixteen the sexual act is a misdemeanour or a felony (*vide* "Rape"), and therefore before sixteen, potency as a possibility need not be considered ; at and above sixteen it is only some other cause than age that can interfere.[1]

In Males. As to the age at which a well-formed male child is capable of erection of the penis nothing need be said : it is a function inherent in the structure of a healthy penis, and a question on the subject cannot arise. At the other end of the scale of life records seem to show that a man of eighty is capable of becoming, and has become a father, and we have no physiological reason for believing that the mere question of "erectio penis" can ever arise on the score of age, apart from disease or accident.

[1] According to the Age of Marriage Act (1929) no person of either sex may be married until he or she has attained the age of sixteen years.

Illness as a cause of Impotency. *In Females.* Again the passive *rôle* played by the female prevents any discussion on this point. A woman who has a natural vagina cannot by any general disease be rendered impotent.

In Males. It is far otherwise with the sex that plays the active part. As a general rule, diseases which do not affect the brain or spinal cord, and which are not attended with great debility, do not prevent intercourse. On the other hand, all diseases which are attended or followed by great general debility must be held to suspend, at any rate temporarily, sexual power on the part of the male; of brain and cord diseases, some are known permanently to destroy potency, while others may, for a time, increase it, though such usually lead to the onset of permanent impotency.

In **acute febrile illnesses** temporary impotency is the rule, especially so long as the fever lasts; when fever is subsiding, or has been replaced by convalescence, the power is rapidly regained.

One of these acute diseases, *viz.*, **Mumps,** must receive special notice. It is well known that mumps has a special proclivity to attack the testicles and such attack, especially in youth and adolescence, is not infrequently followed by atrophy of the gland. After this atrophy, impotence occasionally follows, though admittedly not often; but cases have been reported from time to time. Inflammation of both testicles (double orchitis) is a rare, and according to some authorities, unknown sequel of mumps.

Of **chronic general disease,** *i.e.*, apart from disease of the generative or nervous systems, such as heart disease, chronic nephritis, etc., one can only say that the sexual function is so intimately allied to bodily vigour and healthy nervous energy that the integrity of these may be said to be essential for the integrity of the act; but this must not be taken to mean that we have knowledge that any of these diseases will totally prevent an *occasional* erection which may be used for its natural purpose, though they will undoubtedly diminish the frequency of potency. Habits of drunkenness, the abuse of narcotics, or other drugs, may act in a similar manner, and it is possible that long-continued bad habits of this nature may result in impotency, permanent while the habit lasts, but occasionally disappearing with discontinuance of the habit.

Of **mental and other brain diseases, and of affections of the spinal cord,** many are known to have an effect upon potency in the male subject. Excessive sexual activity is known to be a feature in many forms of mental alienation in their early stages, though this is usually followed by permanent impotency. Lack of sexual power is common in those suffering from paranoia and from dementia præcox and particularly in general paralysis of the insane.

In tabes dorsalis, or locomotor ataxy, interference with the sexual powers in one direction or the other usually occurs. It must not be considered, however, that impotency is a universal and inevitable effect of this disease. Occasionally there is seen in locomotor ataxy the reverse effect, *viz.*, satyriasis or excessive sexual inclination. In a case of myelitis with recovery, potency or impotency would depend upon the situation of

the lesion. Priapism or painful persistent erections of the penis totally unassociated with any sexual desire, which occurs in some lesions, must not be confounded with a potent condition.

These cases of alleged impotency from corporeal disease, when they require to be elucidated by medical evidence, create great difficulty.

A question once arose respecting the legitimacy of a child conceived during wedlock, but born four months after the death of the husband. In presumption of law, the child was legitimate, because husband and wife were at the time living together, and conception and birth were, as to date, in accordance with the ordinary rules. Two months before the supposed date of conception, the husband, a man of intemperate habits, was seized with paralysis (*hemiplegia*) accompanied by coma, and he lost the use of the right side of his body. In about a month he partially recovered, but the paralysis never left him. A month later he was attacked with general dropsy and disease of the liver, and he died five months after the supposed date of conception, and four months before the birth of the child. A year after the death of the husband, the widow married the alleged adulterer, and had by him four children; but for eight years preceding the death of her first husband this woman had borne no child, and it was only when her intimacy with the alleged adulterer commenced, and during her husband's illness, that she became pregnant.

The question submitted to expert medical witnesses was—Was it possible or probable that the husband could have begotten the child in the diseased condition in which he was represented to have been at the date of conception? The opinion given was that it was possible—although highly improbable—because there was opportunity of access, and because sexual power, if lost by the attack of paralysis, might have returned at the material time. Diseases of this kind tended to suspend sexual power; in this particular instance, the effect would have been aggravated by the intemperate habits of the husband, and the general exhaustion and debility from which he was proved to be suffering. Further, the non-procreation of children during the eight years that he was married and in constant habits of intercourse with his wife was clearly not owing to sterility or incapacity on her part, because she had borne children after her second marriage. The evidence regarding the precise bodily condition of the husband about the date of conception was conflicting; and the court decided that the child was the child of the husband. There was no evidence from parental likeness, for the child through whom the claim arose had died some time before proceedings were taken. The legal presumption of legitimacy by wedlock and possible access was too strong to be rebutted by the medical evidence.

It is very rare, if not unknown, for these affections of the spinal cord to have an effect upon the testicles, but should they do so and cause also motor paralysis, there can be no doubt of impotency; but Curling quotes a case from a foreign writer, in which, under paralysis (*paraplegia*), of some years' duration, a man retained sufficient sexual power to have prolific intercourse. When the paralytic person is advanced in age, it is highly probable that he is impotent.

A woman required an order of affiliation on the putative father of her bastard child. She was a widow, and the illicit connection took place about two months before her husband's death. The husband was at the time eighty four years of age; he was bedridden, and for many weeks before his death he could not move in his bed, and was unable to pass his urine without assistance.

The medical opinion of those who had examined him was that he was impotent from physical infirmity, and in this opinion Taylor concurred: stating, however, that unless the male organs were diseased or destroyed, it could not be said that intercourse was impossible. It was, however, wholly improbable that the husband could have been the father of the child.

Blows on the head or spine, by affecting the brain and spinal cord, may produce impotency. Several cases of impotency from this cause are related by Curling. It has been noticed that blows on the under and back part of the head, in the region of the cerebellum, have been followed by loss of sexual power on recovery. Sometimes this is temporary; but at other times, when there is wasting of the testicles, it is permanent and irremediable.

Emotion as a Cause of Impotency. *In the Female.* On account of her passive *rôle*, emotion can hardly be said to be a definite cause of genuine and ineradicable impotency, but at the same time the emotions in a female (especially virgins), may rise to such a height as to constitute practical impotency. This condition is known as vaginismus and may be due either to actual pain on contact or to fear of pain. It may be due to personal aversion or to a general feeling of aversion to the act. It is sometimes the result of homo-sexual perversion. In this condition any attempt at intercourse produces a violent contraction of the constricting muscles of the vaginal orifice and often a contraction of the adductor muscles of the thighs. As a result, intercourse is quite impossible. In many cases the local cause of the condition may be remedied. in others, intercourse under anæsthesia resulting in pregnancy may produce a lasting cure.

In the Male. Emotion is a common cause of temporary impotence, and also of permanent impotency as regards one particular female. Tact and possibly the use of drugs will frequently overcome this form of impotency. The sexual desire, like other animal passions, is subject to great variation; and there are instances on record in which men, otherwise healthy-looking and healthily formed, have experienced no desires of this kind. They are in a state of natural impotency—a condition which the canon law designates as "frigidity of constitution." This is not to be discovered by examination, but rather from their own admission. Under this head we may class hypochondriacal affections.

Hormonic dysfunction may lead to temporary or permanent impotency.

Congenital Conditions

In the male	Non-development of penis. Maldevelopment of penis. Penis adherent to scrotum. Duplex organs (?).
In the female	Absent vagina. Vagina ill developed; too small. Vagina occluded by intra-uterine disease (?).

Acquired Conditions

In the male:
- Disease of penis — Simple. / Malignant.
- Amputation of penis — Intentional. / Accidental.
- Accident to penis.
- Disease of testicles and ducts.
- Excision of testicles.
- Accidents to testicles.

In the female:
- Disease of walls
- Adhesions of walls
- Tough hymen.
- Tumours bulging in the walls.

Causing occlusion of vagina or so much pain as to amount to occlusion.

Some of these conditions are obviously incurable; others may possibly be removable by time, and some by the art of the surgeon.

Deformities or Defects of Development—Congenital Conditions: (*a*) *In Males.* In some instances there is an arrest of development in the external organs, and with this there is generally an absence of sexual desire. Farr met with a case of a man, aged forty-two, in whom the sexual organs remained undeveloped and in an infantile state. There was some difficulty in finding the testicles, in consequence of their small size. On examining the contents of the glands microscopically, no spermatozoa were detected. This person's voice was effeminate, and he was devoid of hair on the chin and pubes. It is not, however, always to be inferred that a male with imperfectly developed organs is incurably impotent.

A gentleman, aged twenty-six, consulted his physician on the propriety of his entering into marriage. His penis and testicles but little exceeded in size those of a boy of eight years of age, and he had never, until this acquaintance with his intended wife, felt the desire of sexual intercourse. He married, and became the father of a family; and at the age of twenty-eight, the organs had attained the full development of those of an adult.

Even the presence of two or three penes, according to Mende, is no bar to the exercise of sexual power, provided one of them possesses the normal features of the male organ. This author refers to cases of duplex organs. One of these sexual monsters, a youth with two distinct penes, was exhibited in London many years ago. He could exercise his functions with either organ, but there was only one testicle to each penis.

An adherent penis is a congenital condition, and as such it may not only in itself be a cause of impotency, but may prevent development of the organ, so that, although the condition may be curable by operation, it is possible that the loosened organ may be of little use.

Finally, the penis may be (for practical purposes), completely absent, owing to imperfect development. Such cases are extremely rare. Incurable impotence is, of course, a result, and probably sterility also, though such is not an absolute necessity.

(*b*) *In Females.* The vagina in females is sometimes absent, a condition which has been known to lead to a curious result, *viz.*, sexual intercourse by means of the urethra. Such a result could not *a priori* be expected, and the condition must be looked upon as incurable impotence. When the vagina is present, but too small owing to deficient growth, the condition may be curable by surgical treatment.

Disease and Accident as Causes of Impotency. Acquired Conditions: (*a*) *In the Male.* (i) *Of the Penis.* Temporary impotence may be caused by an acute disease of the penis, such as gonorrhœa, sores on the glans or foreskin, etc. These need not be further considered; they come under the head of general surgery: more chronic disease, such as epithelioma, could arise only when it had lasted through such a period as to raise a question of the legitimacy of a given child, but each case must be judged on its merits; it is impossible to lay down general rules. Complete amputation of the penis must render a man impotent unless a sufficient length of stump is left to enable introduction to take place.[1]

(ii) *Of the Testicles.* The amount of disease that may exist in one or even both testicles without destroying either sexual appetite or power is remarkable, and even complete destruction or removal does not *at once* destroy the power of connection, though it undoubtedly does so after a time. If the testicles are removed after puberty, the power of erection remains for a long period. For further information surgical manuals must be consulted. Impotency from personal abuse is very doubtful, and depends more on mental emotion than anything else.

(*b*) *In the Female.* Vaginal diphtheria or ulcers of any sort may, on healing, lead to a condition of occluded vagina, in some cases of a permanent and irremediable character. In elderly females a disease known as kraurosis vulvæ has been found to produce the same result. Conditions of toughness of the hymen are curable, and so are many cases of tumours, fibroids, cysts, etc., which may block the canal; prolapse of uterus, vaginal hernia, may be curable. For further information, manuals of gynæcology must be consulted.

Sterility

This is simply the opposite of fertility, or the power of procreation, and as such is absolutely independent of whether or not impotence be present. The distinction between impotence and sterility is very important, and must be clearly borne in mind. Sterility by itself offers no ground for a dissolution of marriage, while impotence may become a just ground. A man or a woman may be sterile and yet not impotent, and impotent yet not sterile. As with impotency, we may classify the causes of sterility thus:—

- General or functional, unconnected directly with the sexual organs.
  - Age.
  - Illness, emotion and unknown conditions.
  - → May lead to or cause temporary or permanent sterility.
- Local or organic, connected directly with the sexual organs.
  - Congenital or acquired conditions.
    - Curable.
    - Doubtfully curable.
    - Incurable.

[1] *Vide* case "Trans Med.-Legal Soc.," vol. 2, p. 114.

Age in Relation to Sterility

In the Male. Puberty. Until the period of puberty the testicles are small, and they increase very little in size in proportion to other parts. Fertile sexual function in the male depends on the proper development of these organs; but the age at which fertility appears differs in different persons. The age of puberty in a healthy male in the British Isles varies from fourteen to seventeen years; its appearance is, however, affected by climate, constitution, the relative development of the associated endocrine glands, and the moral circumstances in which the individual is placed. In some cases it is not fully developed until the age of twenty-one.

The seminal secretion in the male is not prolific until it contains living *spermatozoa*. They are peculiar to the spermatic secretion, and, in healthy males, are always present in it after the age of puberty. In cases in which they are absent, from whatever cause, it is a fair inference that the person is sterile, or that he has lost the power of procreation. A case is recorded in which a man, *æt.* 42, who was married, and whose wife had borne a son then eight years of age, died after four days' illness from strangulated hernia. The testicles, from the fact of their being found in the inguinal canals, were examined, and no spermatozoa were discovered in either of them. These must have been formerly present, although absent at the time of examination, as the child begotten was then eight years of age. During this long interval, the secretion may have undergone a change, and have become infertile.

So long as spermatozoa show movement they are alive and capable (presumably) of fertilising an ovum.

It is certain, then, that a male is incapable of procreating until spermatozoa have appeared in the seminal secretion, and that he loses this power when they disappear. The *age* at which they are formed varies with all the causes that affect puberty.

In the *B.M.J.* for April 23rd, 1887, the following case is reported :—

"A young woman was sent to me for examination, and it was evident she was pregnant. She confessed it, and was brought face to face with her paramour: they both confessed that the woman had led him astray, and allowed him to have intercourse at least a dozen times. The present age of the father is thirteen years and three or four months, and as quickening had taken place at the time of my examination of the woman, the lad could have scarcely attained the full age of thirteen at the time the intercourse took place. The boy, I may say, is well developed as regards the generative organs, but is this not an unusually early age for the development of the procreative functions?"

This is the earliest age at which, in a temperate climate, the procreative power has been reported to be present in the male.

Cases of unusually early development of the external appearances (pubic hair, large penis) of puberty are met with from time to time.

Stone refers to one such in a boy of four, and at the London Hospital in 1907 there was a boy of five who had the pubic hair and penis of an adult. Other cases have been reported from time to time in medical journals.

In a case of contested legitimacy or affiliation, this question regarding the age at which a procreative power appears in the male may have an important bearing on the issue. Thus the person may be so young

as to render it impossible that he should be the father of a child imputed to him. The rule for a medical man to follow on these occasions is this: not to regard the mere *age* of the youth, whether he is above or below the average of puberty, but to observe whether the sexual organs are fully developed, and whether there are about him any of the marks of virility, indicated by muscular development, the growth of a beard, and a change in his voice. If these signs are present, whatever may be his age, there is strong reason to suppose that the sexual functions are developed. We occasionally hear of instances of extraordinary precocity, but the development of sexual power is generally accompanied by other well-marked changes in the person. Sometimes these changes do not make their appearance until after the age of twenty-one.

On the other hand, it may be a question at what time the procreative power disappears in a male. That impotency may possibly occur as one of the natural consequences of *advanced age* is undoubted; but this forms no legal impediment to the marriage of parties, however old. The legal presumption is, that the generative faculty does not disappear through age; and if this be alleged, and legitimacy disputed on this ground, it must be satisfactorily proved by those who would benefit by the allegation. This amounts almost to an impossibility, because it is well known that there is no fixed age at which the sexual functions cease either in the male or female; and individuals, at least of the male sex, who had passed the ages of sixty, seventy, and even eighty years, have been known to be capable of fruitful intercourse. It is believed from anatomical observations on the bodies of aged persons, that the causes of sterility in advanced age are to be found rather in the excretory than in the secretory apparatus. Thus obliterations in the canal of the epididymis and of the vas deferens have been met with, and changes in the vesiculæ, the effect of which is to prevent the accumulation or the passage of the seminal fluid. *Per contra*, spermatozoa have been found in the liquid taken from the testicles of a man upwards of seventy years of age, and on one occasion in the testicles of a person aged eighty-seven. Wagner states that they are to be found in the secretions of men between seventy and eighty years of age. Facts tend to render it highly probable that the fecundating power may be retained by the male up to the age of 100. Dieu has given the results of 105 autopsies of men between the ages of sixty-four and ninety-seven. In 61 per cent. no spermatozoa were found. Four of the cases were nonagenarians: of these none had spermatozoa.

Although the procreative power may be lost at an advanced age, the stimulus for intercourse is often very great, and is often associated with and probably attributable to an enlarged prostate. In all cases of prolonged virility it is observed that the bodily and mental powers are also retained in an extraordinary degree, showing the close relation which exists between the sexual function and corporeal development, even to the last period of life.

The English law on this subject was clearly laid down in the Banbury Peerage case which was brought before the House of Lords in 1806.

Lord and Lady Banbury had been married twenty-one years, without having had issue, when he died at the age of eighty-five years. The peerage was claimed by the descendants of an individual who called himself the son of Lord Banbury; but, in fact, it was alleged that he was the son of Lady Banbury by an adulterer,

during her husband's life. According to the evidence, Lord Banbury did not appear to have been aware of his existence, and the child had always been known by another name.[1]

One of the grounds upon which the legitimacy of the claimant was contested was that the deceased nobleman had become impotent through *age*. The House of Lords decided against the claim, but not on the ground of impotency from age in the husband. It was proved that Lord Banbury was hale and hearty up to the time of his death ; but the moral circumstances of the case, especially the *concealment* of the birth of the child from the husband, were considered sufficient to prove that the child through whom the claim was made was not the offspring of Lord Banbury.

In the Female. Puberty. The following terms represent the relationship between female fertility and menstruation :—

To be fertile a woman must possess an ovary (uterus, etc.).

This ovary must contain living ova capable of development.

These ova attain a certain stage of development, which we may designate ripeness, at successive periods of time.

To these three statements no exceptions can be admitted.

A ripe ovum ruptures its capsule and reaches the uterus.

It is impregnated by a spermatozoon in the uterus.

(To these last two statements certain exceptions occur, proved by the facts of the various forms of ectopic gestation, but these do not invalidate the general truth of the statements).

The escape of a ripe ovum is *generally* associated with certain phenomena, known collectively as menstrual molimina.

The prominent external molimen is an escape of blood from the vagina, which escape is ordinarily spoken of as menstruation.

The escape takes place periodically and commonly every twenty-eight days ; occasionally at longer or shorter intervals.

Observed facts prove that the last three statements have exceptions, and as medical jurisprudence is largely concerned with exceptions to rules, the matter must be considered under the following heads :—

When does menstruation commence ?

Must menstruation precede pregnancy ?

What is the earliest age for pregnancy ?

When does menstruation cease ?

Can a woman become pregnant after the menopause ?

What is the oldest recorded age for pregnancy, apart from the question of menopause ?

When does Menstruation commence ? The menstrual function is commonly established in females in this climate between the ages of *fourteen* and *sixteen* ; but it may occur much earlier—indeed, in some rare instances, a discharge resembling menstruation has been known to occur in mere infants. The occasional appearance of the menstrual flux at an early age does not necessarily imply that in other respects the female attains the development of puberty nor any undue precocity as to sexual instincts. In other cases its appearance has been delayed to a much later period. Perhaps, in this country, the most frequent age for the commencement of menstruation may be taken as fourteen

[1] Sim. and S., 155.

to fifteen years. It is liable to be accelerated in its appearance by certain moral and physical conditions under which a girl may be placed. In warm climates it is commonly asserted that puberty occurs very early, but experience in tropical countries indicates that the maturity of women by no means occurs so early as has been hitherto supposed among Eastern women; the majority of Egyptian girls, for example, menstruate at the age of thirteen years or a little later.[1]

Instances of premature menstruation in the female are numerous, and are far more common than precocious puberty in the male sex. Whitmore met with a case of a female child, who, from a *few days* after her birth, menstruated regularly, at periods of three weeks and two or three days, until she had attained the age of four years, when she died. On inspection after death, she appeared like a much older girl. The breasts were unusually large, and the female organs and lower limbs were considerably developed. The breasts were as healthily developed as in an adult of twenty years, and the sexual organs were also as much developed as in a girl at the age of puberty. The child had the appearance of a little old woman.

R. Hutchison and G. M. Wauhope [2] reported the case of a girl, aged three and a half years, in whom *menstruation began at the age of seventeen months.* There was no family history of precocity. Nothing unusual was noticed until between the tenth and eleventh months, when she had a whitish vaginal discharge which recurred on several occasions till she began to menstruate. The period lasted for two or three days, and returned at regular intervals of twenty-eight days, except on three occasions, when the interval was exactly two months. After her third birthday the intervals became irregular and of longer duration. Pubic hair was noticed at the time of the first period. The breasts were well formed, but no definite date could be given for their enlargement. On abdominal and bimanual examination under an anæsthetic both ovaries were palpable and of normal size. X-ray examination of the skull showed no sign of tumour or other abnormality, but skiagrams of the skeleton showed a bony development usually present about the normal age of puberty. Mentally she was bright but exceedingly obstinate. The voice was harsh and strident. She had no control over her bladder. The cerebro-spinal fluid was normal, and the Wassermann reaction was negative. Precocious puberty is attributable to hypernephroma of the suprarenal cortex, tumours of the pineal gland and tumours or hyperplasia of the sex glands, and it was concluded that the precocity of this child was attributable to over-secretion of the ovaries.

A female child who died at the age of *five* years and six months, attained the height of five feet and a proportionate development of the body throughout. At six months she had cut all the incisor teeth, and at nine months, all the molars. At the eighteenth month the menses first made their appearance, and from that time occurred with great regularity. The hair of the head was long, the breasts prominent, the external genitals well developed but without hair. The pelvis was capacious. The intellectual powers were not more advanced than usual. For similar cases, *vide B.M.J.*, 1, 1901; 1 and 2, 1902.

[1] Elgood, *Jour. Obs. and Gynæcol.*, October, 1909
[2] *Brit. Jour. Child. Dis.*, January—March, 1924, p. 57.

On the other hand, cases of delayed menstruation are from time to time reported. In one case it had not appeared in a married woman, *æt.* 30, who had borne no children, and in another case it appeared for the first time at the age of forty-seven.

Must Menstruation precede Pregnancy? It is extremely rare for impregnation to occur before menstruation has been established, but the possibility of such taking place is generally admitted, inasmuch as it is by no means established what is the precise relationship between the external escape of blood and the escape of a ripe ovum. It is known that true menstruation cannot take place in the absence of ovulation, but the converse does not hold good. There is no question that a temporary condition of amenorrhœa is no bar to conception.

What is the Earliest Age for Pregnancy to occur? It is commonly assumed that as soon as menstruation appears a woman may be considered to have acquired procreative power. It is, however, obvious that in the case of quite young babies such a view is untenable, and even in the cases of young girls it is not common to hear of them becoming impregnated.

A case of pregnancy has been reported [1] in a girl aged six years in which a child weighing 3 kilos was delivered by craniotomy. Secondary sexual characters were observed in the mother at the age of four years.

In another case,[2] that of a girl aged seven years who had never menstruated, the pregnancy went to term and a child weighing 4 lb. 3 oz. was extracted by Cæsarean section.

The following case of parturition in a girl aged thirteen years and eight months is recorded by Dow [3]:—

> At 6.30 one morning I was called to a confinement at a remote farm. I found a girl, aged thirteen years and eight months; beside her on the bed was a fully developed male child, living, and with an evacuated after-birth attached. The vulva was much swollen and œdematous, and protruding from it was a large clot. The uterus was well contracted. Birth had obviously taken place at least one hour before. She had come home from the village school in pain forty hours previously and had lain in bed unattended and terrified till my arrival, ignorant of her pregnancy and the reason for her pain. There was no perineal tear, but some intravaginal sloughing. She and her baby, weighing 6 lb., are recovering normally.
>
> Many years ago, a man, *æt.* 45, was convicted of having had unlawful carnal knowledge of a girl between the ages of ten and twelve years. When intercourse was first had, the girl was *eleven years and eight months old*; it was repeated several times subsequently: and when the girl gave her evidence in court, she was in the last month of her pregnancy: she was then not quite twelve years and six months old. Menstruation had commenced at the age of *ten years and two months*, and had continued regularly up to the time when she first had intercourse with the accused. She was a factory girl, and to the heat, confinement, and association with males, to which factory girls used formerly to be subjected, may be referred the early commencement of puberty.

One of the most notable cases of precocity is that of Anna Mummenthaler, who, as recorded by Haller, menstruated regularly from her second year, and gave birth to a full term child at the age of nine years.

[1] *Zentralblatt f. Gynäk.*, September 23rd, 1933.
[2] *B.M.J.*, September 23rd, 1933.
[3] *B.M.J.*, October 11th, 1924.

When does Menstruation cease (Climacteric or Menopause [1])? The average age at which this function ceases in women is from forty to fifty years; but as it may commence early, so it may continue late in life.

In one case it has been known to cease at the age of twenty-three, and in other instances it has continued to the age of sixty-six and even of seventy-five years.[2] Out of many cases collected by Hogg, the earliest age at which menstruation ceased was twenty-three, the initial period having been sixteen years. In one one woman it ceased at thirty-four, and in two at fifty-three, but in the greatest number (nine) it ceased at forty-seven. Royle described three cases, in two of which menstruation continued up to the age of sixty-seven. Thomas met with a case in which a woman had ceased to menstruate at the age of forty-five, but the discharge suddenly reappeared after an attack of illness when she had reached the age of sixty-nine. The discharge appeared several times, but not with monthly periodicity. It seems that her mother and sister had also menstruated at the ages of sixty-nine and sixty years.

A physician was called to a lady, *æt.* 77, suffering from uterine hæmorrhage. Upon inquiry, he found that she had menstruated monthly up to the time at which he saw her. The discharge lasted from four to five days, and had then left her; but on this occasion it had been very profuse. She was restored by the usual remedies. Other cases are reported. In one of these, a nun, the menses ceased at fifty-two; at the age of sixty-two they reappeared and so continued regularly, until she was last seen at the age of seventy-three. In another instance a nun, aged ninety, had regularly menstruated from the age of fifteen to fifty-two years. The menses then ceased, but reappeared at the age of sixty, without pain, and occurred regularly every month after that date. Her health had been good throughout.

While it may be just to accept these cases of menstruation prolonged much beyond the average, it must not be forgotten that discharges of blood from the vagina, even of a periodical nature, may be due to disease of the parts, polypi, malignant disease, etc.

From these facts, it is clear that it is impossible to fix the age of a woman by the period at which this "change of life" occurs. At the best, it can only be an average of a certain number of instances. This question arose in *Clark* v. *Tatom*, in reference to the identity of a woman, through whom property was claimed by the husband.

The marriage had taken place in 1794; the parties separated in 1809; and the plaintiff's wife, as it was alleged, died in 1843, when, by direction of the defendant, the age of fifty-five was put upon the lid of the coffin. A medical gentleman who attended her in 1841 deposed that, from being then in her menstrual climacteric, he should consider her not to have been more than fifty at that time. He stated that the general period for the cessation of menstruation was forty-four; it was rarely protracted to the age of fifty. On this assumption, it was impossible that the deceased could have been the plaintiff's wife, because at the time of the alleged marriage she would have been only *three years old*. Evidence was given to show that the deceased woman was the plaintiff's wife.

Is it possible for a Woman to become pregnant after Menstruation has Ceased? It is commonly asserted and believed that, after the cessation of menstruation, a woman is sterile. This is doubtless the general rule; but there are many exceptions. Pearson communicated to the *Lancet* the case of a lady aged forty-four, who up to September, 1836, had given birth to nine children. After this the menses appeared only slightly at the regular periods until July, 1838, when they entirely ceased. Owing to this, she supposed that she was not liable to become

[1] An amusing illustration of the ignorance of some insurance offices will be found in the *Lancet*, January 4th, 1902, wherein a legal firm ask where a lady who wished to be insured could get her menopause established or filed!

[2] Whitehead, pp. 145 *et seq.*

pregnant; but on December 31st, 1839—eighteen months after the entire cessation of the menses—she was delivered of her tenth child. Hence conception must have taken place at from eight to nine months after the final cessation of the discharge.

Two or three cases are known of women marrying after the menopause and giving birth to a child at or about the age of forty-nine.

The Oldest Recorded Age for Pregnancy. Numerous instances are on record of females advanced in life bearing children. Duncan concluded, from his researches, that the great majority of the population is recruited from women under forty, but that the mass of women of from thirty to forty years contribute to the general fertility a larger proportional share than the mass of women from twenty to thirty. The age at which women cease to bear children is usually from forty to fifty years; but as they may menstruate, so they may conceive, beyond the last of these periods. Ruttel observed in twelve women that they bore their last children at ages varying from forty-five to fifty years. Ottinger met with an instance of a woman bearing a child at fifty; Cederschjald with another, where the woman was *fifty-three*, and menstruation still continued.

Kennedy records a case in which a woman gave birth to her twenty-second child when she was sixty-three years old, after which she still continued to menstruate.

For a case in which pregnancy and abortion in a woman of fifty-six gave rise to serious trouble, *vide B.M.J.*, 2, 1903, where also other statistics are collected.

In giving a decision in such cases, the law is bound to look to the anomalies connected with the exercise of the generative function; and therefore the limited experience of a few medical witnesses, casually taken, can hardly be expected to supply satisfactory answers to questions of this kind. There is no presumption of law in England respecting the presence or absence of child-bearing power at any period of life; but each case is judged by the whole of the circumstances which attend it.

The oldest authentic case of pregnancy in England occurred in a woman who was born on September 7th, 1852, as certified by the Registrar of North Bierley, and whose child was born on April 19th, 1906, as certified by the Registrar of Knottingley, the certificate thus making the mother's age to be 53 years, 224 days.

Legal Cases with Reference to the Limit of Age for Pregnancy.

Capuron reported a case in France where a man claimed an estate as heir to his mother. His claim was resisted on the ground that, according to the baptismal registry, his mother could not have been the legitimate heiress of the party through whom the claim accrued: because her alleged mother would then have been in her *fifty-eighth* year; and this, it was alleged, was beyond the age of child-bearing. Ancient records were searched, and the claim of legitimacy was admitted, because menstruation and conception had been known to occur at periods of life even later than this.[1] Capuron quotes another case in which a healthy woman menstruated until she had passed her *sixtieth year*, and her last child was born when she was *sixty years* of age.[2]

In *In re Widdow's Trusts*,[3] an order was made for payment out of court of two sums to two ladies respectively. One of the ladies was a widow, about fifty-five

[1] Capuron, "Med. Leg. des Accouch.," p. 93.
[2] *Op. cit.*, p. 98.
[3] L. R. 11 Eq. 408.

years of age ; the other, a spinster, was fifty-three years and eight months old. In both cases the parties were entitled absolutely, subject to the contingency of their having children.

In *Croxton* v. *May*,[1] it was held that where the wife was aged fifty-four years and six months, and had never had any children, her incapacity to bear children was not proved.

In *In re White*,[2] a widow aged fifty-six years and three months, who had only one child (born when she was between twenty-one and twenty-two years of age), and who had lived with her husband for twenty-four years until his death, was presumed to be past child-bearing.

In *In Re Warren's Settlement*,[3] the Court of Appeal refused to relieve a married woman from a restraint on anticipation so as to allow her to deal with her separate estate, although there were no children of the marriage, and the husband was fifty-four and the wife fifty-five years of age. The court declined to assume that the wife was past child-bearing.

General Causes of Sterility

(*a*) *In the Male.* Apart from impotence, the law takes no cognisance of sterility in the male : as a medical question it could only be decided by proving by microscopic examination that spermatozoa are absent from the testicular secretion or, if present, that they are not in a normal living state. Personal abuse is alleged as a cause, but this is extremely doubtful. No further comments are necessary from a medico-legal point of view.

(*b*) *In the Female.* We have only to appeal to known facts amongst domestic animals and amongst wild animals kept in a state of confinement to be certain that the environment and nutrition have some influence upon conception ; many infertile animals have been examined after death and found to be healthy and capable of bearing progeny. Again, in human beings there are many instances of women who have been sterile to one husband and fertile to another without any suspicion of the fault lying with the husband, or rather, without any proof that such was the case. The explanation of these facts is obscure. On the one hand, disease may be of such a subtle or physiological character that the ova of the female may be incapable of fertilisation ; on the other hand, there may be mechanical impediments to the proper meeting of sperm and ovum, impediments of such slight character as to be overlooked on autopsy. For a few further remarks, *vide* " Sterility from Disease."

Local Causes of Sterility

Congenital Conditions

In the male
- Absence of testicles.
- Absence of penis (?).
- Maldevelopment of testicles.
- Disease of testicles *in utero* (?).
- Misplacement of testicles (?).
- Malformations of penis, epi- and hypospadias.

In the female
- Absence of ovaries.
- Absence of uterus.
- Maldevelopment of ovaries.
- Maldevelopment of uterus.
- Absent vagina.

[1] 9 Ch. D. 388.
[2] [1901] 1 Ch. 570.
[3] W. N. (1883), 125

Acquired Conditions

In the male	Complete amputation of penis (?). Excision of testicles. Disease of testicles. Accident to testicles. Atrophy of testicles from mumps, etc. Masturbation and spermatorrhœa.
In the female	Excision of ovaries. Disease of ovaries. Excision of uterus. Disease of uterus. Occlusion of vagina from disease. Disease of vagina.

Congenital Conditions in the Male

It will only be necessary to say a few words on some of these conditions.

Complete absence of testicles and of penis are theoretically possible conditions, but no record seems to exist of a case in which the fact has been of any medico-legal interest; and, except in monstrosities, such a condition must be of most extreme rarity. Of maldevelopment of the testicles, due to intra-uterine disease, the same remarks may be made; a case is quoted under "Impotence," *supra*.

Sterility in Relation to Monorchism and Cryptorchism. In some males the testicles do not descend into the scrotum at the usual period, but one or both may remain in the abdomen, or in the inguinal canals, and descend some time after birth or not at all; or one may be found in the scrotum, and the other remain during life in the abdomen. Persons with no apparent testicles are called *Cryptorchids*, while those who have only one testicle apparent are called *Monorchids*.

When one testicle only has descended, there is no ground, *cæteris paribus*, to impute sterility: the descended organ has been found healthy and to contain spermatozoa, while the retained testicle and its ducts have in some cases been found not to contain spermatozoa.

It has been stated that in all cases of non-descent, the testicles are congenitally defective, and further, that the persons, although capable of sexual intercourse, are incurably sterile. Hunter thought so. Some cases support this view, for spermatozoa have at times not been found in an undescended testis. But it is now known that a cryptorchid is not *ipso facto* sterile. Some are and some are not: and the question therefore is in no need of discussion as to rules or principles. Each case must be determined by other evidence than that of general medical knowledge, *i.e.*, on its merits, as in the following case:—

"W. H., a seaman, *æt.* 34, admitted for strangulated hernia. At the operation the right testicle was found behind the loop of strangulated gut in the inguinal canal, the left was easily felt at the margin of the external ring in the canal; recovery was rapid, but he had an attack of orchitis. The earlier history is as follows: Since childhood has always enjoyed excellent health. The testicles were never at any time in the scrotum, and of this he never seems to have taken any particular notice. He is now thirty-four, a strong, fully developed muscular man. Since puberty has had full sexual power, and was married sixteen years ago at the age

of eighteen; five children born to him, one being now alive. The testicle which was seen and examined in the canal was smaller than normal, soft and elastic on pressure. The left appears of about the same size and character. The penis was well developed. I may say that the man has been seen by several practitioners, and there is no doubt whatever of the paternity of the children, his wife being devoted to him and her children, and the home life is a particularly happy one.

As a physiological fact, the organs which have not descended are not always defective in structure or function. The facts above mentioned prove that there is no reasonable ground for pronouncing a man to be sterile, merely because his testicles are not in the scrotum. If, with a non-descent of these organs, there should be a non-development of the other external organs, and this is accompanied by a total want of the features of virility, then the person may be impotent or sterile, or both. The testicles may, in such a case, be either congenitally absent or physically imperfect—a fact only ascertainable by an examination of the body after death. On the other hand, in cases in which there are no external marks of effeminacy, or other grounds for suspecting a want of procreative power, and the person is capable of sexual intercourse, this imperfection does not offer any bar to marriage, nor is it a sufficient ground for divorce. It would not justify a medical man in denying the paternity of a child on a question of affiliation, bastardy, or inheritance; and so long as the power of sexual intercourse existed, it would not justify him in pronouncing the person to be incurably sterile. The capacity for sexual intercourse is the sole fact to which the English law looks on these occasions.

Epi- and Hypospadias. These terms refer to malformations of the penis of such a nature that the opening of the urethra—through which the seminal fluid has to pass—is not situated at the end of the glans penis, but at a varying distance from the end, either on the dorsum (epispadias) or on the under surface (hypospadias) of the penis. Cases of either condition are by no means infrequent. Unless the condition is associated with some other developmental anomaly—absence or ill development of the testicles—there can arise no question of *absolute* sterility, for the seminal fluid is in itself fertile. The question is whether, in the natural act of copulation, the semen will be placed within the vaginal orifice, and so have an opportunity of reaching the ovum (*vide* "Artificial Impregnation"). It is reported that some aboriginal tribes have recourse to artificial hypospadism as a means of allowing unfruitful connection.

The power to have fruitful intercourse will in either case depend on the situation of the urethral orifice.

Where the urine can pass, the seminal fluid can pass; and the only question is, whether the intromission can be such as that the misplaced orifice should come in contact with any part of the vagina. This must depend on the situation of the orifice. Similar remarks apply to epispadias. These malformations are sometimes remediable by operation; but whether remediable or not, they are not, in any circumstances, to be regarded as absolute causes of sterility.

Congenital Conditions in the Female

The congenital conditions enumerated above require little discussion. Sterility rarely becomes a medical question in contested cases of

legitimacy ; for the claim on the part of a person to be the offspring of a particular woman, unless she were in collusion with the claimant, could only be made after her death ; and if not disproved by medical evidence, showing that the woman could not have borne children, it would in general be easily set aside by circumstances. If the uterus, ovaries, or other parts were congenitally defective or absent, or if there were external sexual malformation, accompanied by occlusion or obliteration of the vagina, a medical witness would have no difficulty in saying that the woman must have been sterile. A mere occlusion of the vagina, removable by operation, does not necessarily indicate sterility, for the internal organs, including the womb, may be healthy and sound. In some instances, the ovaries or the uterus are entirely absent, or the Fallopian tubes are obliterated—conditions which cannot in all cases be determined during life ; whilst in other instances these organs exist, but are defectively developed.

The absence of a womb, and the absence of the function of menstruation, do not necessarily prevent the development of strong sexual propensities, although there is of course incurable sterility. A congenital absence of the uterus and ovaries is not inconsistent with a full development of other parts.

A woman, *æt.* 40, had enjoyed good health up to the last year of her life. On inspection there was a complete absence of the uterus and ovaries. The vagina was normal, terminating in a *cul de sac*. The clitoris was well developed, together with the labia and mons veneris. The breasts were large and plump. The whole aspect attested the attributes of a well-formed woman.

Some of these deficiencies can be detected only after death.

Some of the external physical causes of sterility in a woman are removable by art. Thus, when the vagina is unnaturally closed, this condition may be often remedied by operation. In an instance of this kind, the woman subsequently married and bore a child. If the internal organs are in their normal conditions, the slightest aperture will suffice for impregnation. Penetration is not necessary (*vide* "Signs of Virginity"). Women have thus been known to conceive in circumstances which appear quite adverse to the possibility of conception : and when they have arrived at the full time it has been found necessary to make a free incision into the parts which resisted the passage of the child's head. A remarkable case of this kind is quoted in the *B.M.J.*, 1, 1910, and there are many others of a similar nature on record. Sometimes the external passage is free, but the congenital occlusion may be at the mouth of the uterus. This is a cause of sterility, which, however, admits of remedy by operation.

Acquired Conditions

In the Male. With regard to penile conditions, the question is one of impotency rather than of sterility, but it is obvious that complete amputation must render both conditions certain.

With regard to testicular disease, accident, or removal, the question is more complicated, for it involves two separate considerations, or perhaps three. First, the actual formation of spermatozoa ; secondly, whether they are formed in a living state ; and thirdly, whether they can escape from the male organ. There can be no doubt that

amputation of the testicles causes *ultimate* sterility, though even here animals have been known to be prolific for a certain time after castration ; and one case is on record in which a man, both of whose testicles had been carried off by gunshot, is said to have retained the power of impregnating his wife, after the healing of the wound. It must be assumed that the vesiculæ seminales contained sufficient semen for the purpose.

With regard to disease, it is by no means easy to determine what degree of it will render a man sterile ; if with extreme wasting of the testicles or very extensive disease, sexual desire entirely disappears, there can be little doubt of sterility, but the absence of desire in such circumstances cannot be proved by medical evidence, and must depend upon the statement of an interested party. It is stated to have been proved by microscopical examination that spermatic secretion of living spermatozoa continues even when only a small part of the gland has remained healthy.

Stricture of the urethra need not be considered, for where urine can pass semen can do so, but it is otherwise when the spermatic cords are, from operation or disease, blocked, ligatured, or cut in any part of their course ; such a condition renders sterility certain.

It is alleged that excessive personal abuse will render a man sterile ; it is difficult to obtain evidence on this point, but the statement is incorrect, though a condition of emotional impotency may probably temporarily arise from this cause.

In the Female. This subject belongs to clinical gynæcology rather than to forensic medicine, and little needs to be said upon it. Cases are recorded from time to time in which women have borne children even after double ovariotomy. Clinically there can be no doubt that the diseases enumerated above cause an incapability on the part of a woman of having *living* children, but as this is not considered sufficient ground for divorce, it never comes before a court of law as a subject upon which medical evidence is required *re* Sterility, though the *cause* of the disease may do so (*vide* " Divorce ").

Cases have occurred of pregnancy in women the subjects of bilateral ovarian dermoid tumours. This is quite comparable to local disease of the testicles.

An absence of the menstrual function (amenorrhœa) has been described as a cause of sterility ; but several cases have been reported which show that women who have never menstruated, or in whom the discharge has appeared and has ceased for many years, and who are otherwise healthy and well formed, may become impregnated. When, however, the absence of menstruation depends on a closure of the mouth of the uterus, or other physical causes of the like nature, there will of course be sterility. If in other respects a woman is well formed, she cannot be regarded as in a necessarily incurable condition.

Women who have not menstruated before marriage have conceived immediately after their marriage. Instances are well known to occur in which a woman has not menstruated for some months previous to conception and thus gestation has appeared to be considerably protracted. Hence a woman may conceive, although menstruation has not commenced, and although it may have commenced and afterwards ceased (*vide ante*, under " Age and Sterility ").

There is a popular notion that women during menstruation and lactation are sterile, but this is incorrect. Well-developed and healthy women remain sometimes married for years without having children, then, without any apparent change of habit, they become impregnated, even after a barrenness of fifteen or twenty years. Any diseased condition of the system is unfavourable to impregnation, and *a fortiori* diseases affecting the uterus or ovaries.

For the effect of chronic uterine disease on fertility, manuals of gynæcology must be consulted.

On the whole, the physical and irremediable causes of sterility in the female are not so apparent as in the male, because in the former the generative apparatus is placed internally, and slight changes in its various parts, sufficient to produce permanent sterility, cannot be determined by an examination during life.

The whole subject is one in which it is extremely important to note the difference between fact and presumption. Sterility and pregnancy are facts while they last, but to state that sterility must be permanent is an assumption which must be supported by strong evidence before it can be accepted as a fact.

CHAPTER II

VIRGINITY AND DEFLORATION

Signs of Virginity

A *virgo intacta* was once defined at a critical moment by a judge as a *rara avis*, and, so far as medical evidence is concerned, the definition might almost stand, for though many conditions have been found in *virgines intactæ*, there is no one of them that may not be found in a woman who has had isolated acts of connection, or even habitual connection, provided that she has not borne a child. The question may assume some importance in divorce cases and in cases where the chastity of a woman is involved. The following are the points usually relied upon to establish the condition of *virgo intacta*.

The Breasts. These in young adults are commonly firm and hemispherical, the nipples small and surrounded by areolæ from light pink through slightly darker shades of colour, according as the possessor is blonde or brunette. It cannot be supposed that a single act of coitus will alter this, and occasionally the breasts of women who have borne children conform to the above description. It is, therefore, evident that the breasts are of little value as a sign of virginity.

The Hymen. This is practically always present in a *virgo intacta* in some form or other, but in certain cases, rare though they may be, it is congenitally absent. The hymen may be intact, but this does not prove non-intercourse, because women have been known to conceive with the hymen uninjured ; and an operation for a division of this membrane has been frequently rendered necessary before delivery could take place.

The explanation of this fact lies in the (1) form, (2) structure and consistency of the hymen, and in (3) the nature of the opening or openings in it. For full particulars, works on gynæcology must be consulted. Here it is sufficient to state that—

(1) The form may be circular or crescentic, *i.e.*, entirely or only partially attached to the whole circumference of the vulval aperture.

(2) The structure and consistency may be that of firm strongly resistant connective tissue with many blood vessels, etc., or that of a very thin elastic yielding membrane with scarcely any blood vessels in it.[1]

(3) The opening may be single or multiple, like a colander, large or small, with a thin or thick ring of tissue round it, yielding and elastic or firm and resistant. Again, the hymen, whatever its original structure,

[1] Von Hofmann's Atlas, E. Tr.; Figures 8 to 27.

nature and extent, may be destroyed by ulceration as a result of inflammation or by accident. When the membrane has been thus destroyed by disease or other causes, or when it is congenitally absent, a medical opinion, based on the condition of the hymen, must be more or less conjectural; for one intercourse could hardly so affect the capacity of the vagina as to render the fact evident through life, and there is no other datum upon which an opinion could be based. If a fairly resistant hymen with small opening be found quite intact, there is a fair presumption of chastity; if, on the other hand, there be a very soft and resilient hymen with a large opening, no opinion can be offered. The presence of the hymen is of course incompatible with the assumption that the woman has borne a child.

So long as there are facts which show that women have actually conceived with the hymen still in its normal state, it is inconsistent to apply the term *virgo intacta* to women merely because this membrane is found entire. A woman may assuredly have an unruptured hymen and yet not be a *virgo intacta*. This can be decided only by the special circumstances proved in each case.

Dixon Mann makes the following observations on a ruptured hymen :—

"The hymen may be ruptured by an adequate force of any kind, apart from sexual intercourse. It is reported to have given way from the presence of blood-clots during menstruation, from ulceration following diphtheria or other diseases, from jumping, riding on horseback, or falls on a hard projection. Masturbation has been stated, but probably without sufficient grounds, to be a cause of rupture of the hymen; in the majority of cases of habitual masturbation, the hymen will be found intact, the manipulations being limited to the parts anterior to it. Medical examination or applications may cause injury to the hymen. Some of these reasons for the absence of, or injury to, the hymen are quite feasible, others are far-fetched; each case has to be judged on its own merits."

Several cases have been observed in which little girls have had their perineum torn from vagina to anus, opening the whole rectovaginal septum, by sliding down a baluster and striking the flat boss on the top of the newel-post.

The Vagina. In girls who have not had intercourse, the vaginal walls are rugose and firm. This is true, but again it can hardly be supposed that one act will so stretch them as to deprive them of tone and rugæ, but if the act be habitual and, *a fortiori*, if parturition has taken place, these rugæ are likely to have disappeared. For all that the sign is likely to fail when most wanted, for in habitual connection there is likely to be other evidence.

Fourchette and Perineum. The former is a thin posterior edge of the margin of the mucous membrane, the latter the edge of the skin, limiting the vaginal orifice. In a *virgo intacta*, both of them are discernible and untouched. The fourchette is frequently ruptured by the first connection. The relative sizes of the aperture and the organ introduced through it, together with the degree of violence of introduction, would account for either membrane being torn or remaining intact.

Signs of Loss of Virginity

Little can be added to what has been said above on the presence of virginity. In cases of alleged rape (*vide* " Rape "), the presence of signs of violence, tears, signs of inflammation, discharges, are all of material assistance, but these are quite apart from the abstract question of whether or not the person in question is a *virgo intacta* or not. We can only say that the absence of the signs mentioned above constitutes the only possible chance of answering the question, if the woman be examined at a time when signs of recent defloration have had time to disappear.

CHAPTER III

PREGNANCY

The Legal Position of Pregnancy.
The Signs of Pregnancy.
Superfœtation.
The Duration of Pregnancy.

There are several reasons for which a medical man may be requested (he cannot be ordered ; at least the order has no validity) by the law to examine a woman to determine whether she be pregnant, *viz.* :

Where a woman convicted of capital crime alleges that she is pregnant.

Succession on intestacy.

To assess damages in a seduction case.

He may also find it necessary in private practice to do so—

To disprove a libel.

To suggest the advisability of a marriage.

For many other purposes in his practice.

We may explain some of the legal purposes before entering upon the details of the evidence.

LEGAL CASES IN WHICH THE ESTABLISHMENT OF PREGNANCY IS REQUIRED

Sentence of Death is not passed on Pregnant Women

In England, by the Sentence of Death (Expectant Mothers) Act, 1931, sentence of death cannot be passed on a pregnant woman. The trial jury, without being re-sworn, is under a duty to determine whether or not a woman convicted of murder (or of any other crime punishable by death) is pregnant. If the jury finds, upon hearing medical evidence, that the woman is pregnant, she will be sentenced to penal servitude for life instead of to death. The woman may appeal to the Court of Criminal Appeal against a finding that she is not pregnant. The ancient right formerly possessed by a woman to move in arrest of execution has been abolished.

Succession on Intestacy

Prior to the Administration of Estates Act, 1925, it was possible for the heir-at-law to obtain a writ to require some proof of the statement made by a widow that at the date of her husband's death she was pregnant ; but inasmuch as the old law of inheritance has been abrogated by the new statute, the old practice has ceased to be of importance. The heir-at-law has been abolished by the said Act.

Pregnancy may be feigned or alleged, and it is a medical man's business to be able to detect such imposture. A woman who is an impostor usually feigns an advanced stage of pregnancy. Although she may state that she has some of the symptoms depending upon pregnancy (and, unless she has already borne children, she will not even be able to sustain a cross-examination respecting these), yet it is not possible for her to simulate without detection a distension of the abdomen or the state of the breasts. If she submits to an examination, the imposition must be detected; if she refuses, the inference will be that she is an impostor. Women have been known to possess the power of giving apparent prominence to the abdomen, and even of simulating the movements of a child by the aid of the abdominal muscles. By placing them under the influence of chloroform, the abdomen at once collapses, and the imposture is detected. These cases of spurious pregnancy are sometimes met with in hysterical females. "Phantom tumours" of the abdomen are now well known in practice.

That pregnancy may take place from intercourse during unconsciousness on the woman's part, or after rape, *i.e.*, involuntary connection, is, of course, possible.

On the other hand, **it is quite possible for women to be pregnant and not to know it.** It is hardly necessary for examples to be given, for such cases are the everyday experience of most medical men.

A woman, thirty years of age, had three children, aged three, four, and five. Her three pregnancies and confinements had been perfectly normal, and there was no history of any gynæcological complaint. She woke one morning to find the sheets soaked with a clear and copious vaginal discharge, and twenty-four hours later she was delivered of a living child weighing about five and a half pounds, labour being associated with the expulsion of a considerable excess of amniotic fluid. Inquiry into her history revealed that she had menstruated regularly each month for the usual number of days, that she had felt no movements, nor had she suffered from any malaise or digestive disturbance. The larger size of her abdomen had not attracted her particular attention, since previous pregnancies had left the walls lax, and she attributed any swelling to her habitual constipation. Her breasts had shown no alteration from the state in which they had been left by previous pregnancies. A physician examined the patient about twenty hours before delivery, and he found a uterus enlarged to the size expected at about five or six months, with very exaggerated ballottement of a fœtus, no movements of which could be felt, nor could any fœtal heart sounds be heard.

The following is an extreme case :—

A married woman who had not had a child for nineteen years found herself, as she thought, getting unusually stout. She was moving about with her family to different places. At last her size alarmed her, and she thought she was suffering from dropsy; she consulted a physician, who informed her that she was in an advanced state of pregnancy. She treated this opinion with great contempt. She and her daughter arrived at a miserable inn; on the night of their arrival this lady was seized with the pains of labour, and was delivered of a child. She had made no preparation for the birth, and up to the moment when she was seized with labour pains she had not, with all her former experience, the slightest belief that she was pregnant.

Instances of this kind are important in reference to alleged unconscious delivery in the cases of women charged with infanticide. At the same time, many of the cases in which there are motives for pleading unconscious intercourse or pregnancy require close examination: they will frequently be found to be quite unworthy of belief.

Concealment of Pregnancy. By Scots law, if a woman conceals her pregnancy during the whole period thereof, and if the child of which she was pregnant be found dead, or is missing, she is guilty of an offence, and is liable to prosecution. The evidence which is relied on for conviction is clear and distinct evidence of the actual delivery of a child. This is usually furnished by medical witnesses. Scots law, by making the concealment of pregnancy in the circumstances above mentioned, an offence, proceeds on the principle that every pregnant woman is bound to make preparations for the safe delivery of a child; and it is therefore assumed that if a child is born clandestinely, without preparation, and is found dead or is missing, its death is owing to the lack of such preparation.

Signs of Pregnancy in the Dead. There is no special case in law wherein the *fact of pregnancy* requires to be verified after the *death* of a woman; but an examination may be necessary to determine the identity of a body, or to rescue the reputation of a woman from a charge of unchastity. The discovery of an embryo or fœtus with its membranes in the uterus, or any other product of conception, such as a hydatid mole or lithopædion, would, of course, at once solve the question, should the necessity for an examination occur; and the practitioner will remember that, even supposing many years to have elapsed since interment, and the body to have been reduced to a skeleton, still if the fœtus had reached the period at which ossification takes place, traces of its bones may be found amidst the bones of the woman by direct observation or by means of an X-ray examination of the material taken from the pelvis. In examining the body of a woman long after death for the purpose of determining whether she was or was not pregnant at the time of death, it may be borne in mind that the unimpregnated uterus undergoes decomposition much more slowly than other soft organs.

In the case of a woman who had been missing for a period of nine months—whose body was found in the soil of a privy, so decomposed that the bones separated from the soft parts—the uterus was of a reddish colour, hard when felt, and its substance firm when cut. The fact was of importance. It was alleged that the deceased was pregnant by a young man, and that in order to conceal her condition he had murdered her. From the state of the uterus, it could be affirmed that this organ was in its virgin condition, and that the deceased was not pregnant at the time of her death.

It may happen that the appearances in the uterus are sufficient to create a strong suspicion that the woman had been pregnant, but the ovum, embryo, or fœtus may have been expelled; in such cases a microscopic examination of the uterus, ovary, etc., may show the presence of certain of the products of conception or a condition of the mucous membranes and muscular tissues indicating the fact of pregnancy.

In cases of recent pregnancy the site of the placenta may afford valuable evidence.

MEDICAL EVIDENCE OF PREGNANCY

The signs of pregnancy from a medical point of view are commonly divided into the presumptive or probable and the certain, and possibly this is the best way to consider them. For further details text books on midwifery should be consulted.

Presumptive or Probable Signs of Pregnancy. *Cessation of Menstruation.* It is well known that in the greater number of healthy females the menses cease when conception has taken place, but, on the one hand, women who have never menstruated have been known to become pregnant, and, on the other, women have been reported to have menstruated during pregnancy (see case, p. 25). Theoretically menstruation is possible during the first three months of pregnancy, but true menstruation appears to be impossible after that period owing to the fusion of the decidua vera and the decidua capsularis. Bleeding at later periods is probably due to other causes. Again, either menstruation or its suppression may be feigned, for in either case the woman probably has strong motives for asserting that which is not. To discuss the numerous conditions besides pregnancy which may cause the menses to cease would be out of place here, but will be found in any standard work on midwifery or gynæcology.

Morning Sickness, although a frequent phenomenon in the early stages of pregnancy, is so frequently caused by other conditions (alcohol, for example) that it may be dismissed without further notice.

Changes in the Breasts. These need not be described in full, nor criticised; they essentially consist in increased growth with increased physiological activity. There are pigmentary changes consisting of a darkening in the colour around the nipple about the second month, and in and around the areola a number of raised spots known as Montgomery's tubercules make their appearance. Colostrum is found, about the third month. The nipple increases in size and darkens in colour, and about the fifth month the formation of a secondary areola may be observed. Secretion of milk may occur in the non-pregnant female and has been known to occur in children and in males.

Progressive Enlargement of the Abdomen. This, like the foregoing, is *primâ facie* suggestive of pregnancy, but may be due to other causes. It is sufficient to say that any well-educated medical man could hardly fail to differentiate a pregnant uterus from other causes of a prominent abdomen, especially if an anæsthetic were given before examination. At the same time it must be admitted that mistakes have been made in clinical practice, so that care is needed in the differential diagnosis.

Quickening. Quickening is the name applied to the peculiar sensations experienced by a woman about the fourth month of pregnancy. The symptoms are popularly ascribed to the first perception of the movements of the fœtus, which occur when the womb begins to rise out of the pelvis; and to these movements, as well as to a change of position in the womb, the sensation is probably due. The movements of the fœtus are perceptible to the mother before they are made evident by an external examination.

In olden days quickening was legally important, because the law demanded proof that a woman was "quick with child." This is no longer the case, and we need only mention here that the period at which it occurs is very variable in a genuine pregnancy, and that women practise much self-deception about its occurrence in cases of spurious pregnancy.

Vaginal Softening, discolouration and pulsation are constantly found in the early months of pregnancy.

Softening of the Os is a constant feature in pregnancy, as is a *progressive softening of the cervix* or of the lower part of the body of the uterus which is detectable by bi-manual examination about the eighth to tenth week when the uterus is beginning to rise out of the pelvis. This forms the basis of Hegar's sign.

Uterine Souffle. A blowing sound heard just over the pubes, by means of the stethoscope; it is constant in pregnancy, but is also heard in other conditions. It is synchronous with the maternal pulse.

Ballottement. A useful test for pregnancy is the shock perceptible to the finger on giving a sudden impulse to the child through the neck of the uterus. The child floating in the liquor amnii is driven by the impulse against the other side of the uterus, and it is this blow against the womb that is perceptible to the hand placed on the abdomen.

Rhythmical Contractions and Relaxations of the Uterus. These are not always perceptible, but when felt are strongly suggestive of pregnancy. It is seldom that five or ten minutes elapse without these contractions being perceptible to the hand resting on the abdomen. At one time a tumour is plainly defined, more or less firm, and resisting; in a short time this becomes flabby, and sometimes not to be found: again the uterus contracts, and the tumour becomes as apparent as before. They sometimes occur in soft fibroid tumours.

Certain Signs of Pregnancy. These are all characteristic of the latter half of pregnancy.

Movements of the Child. These are not always to be appreciated, but they cannot, when they occur, be mistaken for anything else, and are indisputable evidence of a child, living at the time of examination.

Recognition of the Fœtal Parts by abdominal palpation renders the diagnosis certain.

Sounds of the Fœtal Heart. By the application of the ear or a stethoscope to the abdomen, at or about the fifth month of pregnancy (rarely earlier), the pulsations of the fœtal heart may be recognised and counted. These pulsations are not synchronous with those of the arteries of the mother; they are much more rapid, and thus with care it is impossible to mistake them. Their frequency is in an inverse ratio to the state of gestation, being usually 160 at the fifth, and 120 at the ninth month. Rarely, however, the fœtal pulse may descend to 80 or even 60 beats per minute. This sign, when present, not only establishes the fact of pregnancy beyond all dispute, but shows that the child is living. The sound of the fœtal heart is, however, not always perceptible: when the child is dead, of course it will not be met with; but its absence is no proof of the death of the child, because the hearing of the pulsations by an examiner will depend very much upon the position of the child's body, the quantity of liquor amnii, and other circumstances. Thus the sounds may be distinctly heard at one time, and not at another: they may be absent for a week or fortnight, and then will reappear: so that, although their presence affords the strongest affirmative evidence, their absence furnishes uncertain negative

evidence: and several examinations should be made in the latter case before an opinion is formed. The reason why the sounds of the fœtal heart are not always perceived is owing not only to change in the position of the child, but to the vibrations having to traverse the liquor amnii and the soft layers of the walls of the abdomen. The presence of much fat in these layers intercepts them. The point where the sounds can be most readily perceived is commonly in the centre of a line drawn from the navel to the anterior superior spinous process of the ilium on either side—perhaps most commonly on the left.

As all certain signs refer to an advanced stage, a witness may be asked, What are the unequivocal indications of pregnancy *before the fifth and sixth months*? The answer to this question is of little moment to a medical jurist, since he is rarely required to give an opinion at so early a period. In all *legal* cases, when pregnancy is alleged or suspected, it is the practice for a judge or magistrate, on a representation being made by a medical witness, to postpone the decision one, two, or three months, according to the time required for obtaining *certain evidence*. The most experienced practitioners agree that before the *sixth month* the changes in the neck and mouth of the uterus are of themselves too uncertain to enable an examiner to form a certain opinion from one examination only; and, *a fortiori*, it is impossible to trust to external signs alone. A skilled gynæcologist may be able, even by the end of the second month, to give a decided opinion one way or the other, if he have repeated opportunities for examination at intervals; but as most practitioners do not belong to this class, it is well to state plainly that only a problematical opinion can be given, and to ask for an adjournment of the case.

The x-ray Diagnosis of Pregnancy. Under favourable conditions the presence of a fœtus may be diagnosed by X-ray examination as early as the middle of the fourth month, and each succeeding week renders the diagnosis more certain and easy. The rays are also valuable in the diagnosis of plural pregnancy, malformations of the fœtus and death of the fœtus.

Biological Tests for Pregnancy. In the female subject certain hormones are normally excreted in the urine during the whole period of sexual maturity. During pregnancy the excretion of these substances is enormously increased, and their identification in the urine of the subject has been used for the diagnosis of pregnancy.

The hormones are œstrin, or the female sex hormone, and the gonadotropic hormones normally secreted by the pituitary gland. The onset of gestation is associated with an immediate and marked increase in the elimination of the gonadotropic hormones, whereas the excretion of œstrin rises comparatively slowly. Hence the method of choice in the diagnosis of pregnancy is dependent upon the detection of the gonad stimulating hormones.

Three methods are in use: The Aschheim-Zondek Test, The Friedman Test, and the Xenopus Test.

The Aschheim-Zondek Test. In this test immature female mice are injected with the morning urine of the patient. After 100 hours the animals are killed, and the ovaries examined. In a positive case the ovaries are enlarged and show evidence of blood spots to the naked

eye. According to different observers, this test has a reliability of 97 to 99 per cent.

The Friedman Test. This is a modification of the above test and has about the same reliability. It is rather more simple, one intravenous injection of 10 c.c. of the morning urine being made into the ear vein of an immature female rabbit. Ordinarily the ovaries of an isolated unmated rabbit contain no corpora lutea or hæmorrhagic spots, since the rabbit does not ovulate spontaneously but only after coitus. The injection of the urine of a pregnant woman stimulates the activity of the ovary, and corpora lutea and hæmorrhagic spots can be observed within twenty-four to thirty-six hours.

The Xenopus Test. Prepared urine from the suspected case is injected into *xenopus laevis*, an African species of toad. This appears to be a rapid and satisfactory test, in circumstances in which the toads can be kept under proper conditions. If pregnancy is present the animal ovulates very rapidly and no killing is necessary.[1, 2]

These serological tests are available within about twelve to fifteen days of impregnation and form a valuable aid to the diagnosis of pregnancy at an early stage. The test remains positive until some days after the termination of pregnancy or of the death of the fœtus. It is positive in chorio-epithelioma and in hydatid mole.

THE DURATION OF PREGNANCY

This will be discussed under three headings :—

1. What is the average period of gestation ?
2. To what extent can this period be shortened and yet a viable child be born ?
3. To what extent can gestation be prolonged ?

Before discussing these in full it is well to refer briefly to the unusual forms of pregnancy known as **superfecundation** and **superfœtation.**

Superfecundation is the fertilising by separate acts of coitus of two ova which have escaped at the same act of ovulation ; the possibility of such an occurrence is admitted by most authorities.

Superfœtation is the fertilising of two ova which have escaped at different acts of ovulation. Up to the third month, when the decidua vera unites with the decidua reflexa, we must admit that it is a physical possibility for spermatozoa to reach a second ovum, but the doubt is whether ovulation is suspended so soon as one ovum is impregnated, *i.e.*, during the whole time of pregnancy. Authorities are much divided on the subject, but there is a general disbelief in the possibility of superfœtation. Cases in which a second child has been born a considerable time after the first are usually explained on the assumption that there has been a twin pregnancy, in which the second child has been abnormally slow in development and has lagged behind the first. After the birth of the first child the second then obtains its proper nourishment and becomes fully developed in a month or two and is born a full time child. Difference in development of uniovular twins is common. Cases

[1] Elkan, E. R., *Brit. Med. Journ.*, 2 : 1253 (Dec. 17th, 1938).
[2] Crew, F. A. E., *Ibid.*, 1 : 766–770 (April 15th, 1939).

have been published, however, which support the view that superfœtation can actually take place.[1, 2, 3]

The following appears to be a case wherein a medical opinion might be required respecting this alleged phenomenon.

A married woman, six months after the absence or death of her husband, gives birth to an apparently mature child, which dies: three months afterwards, and nine months after the absence or death of her husband, she may allege that she has given birth to another child, also mature.

A question may arise, whether two mature children could be so born that the birth of one should follow three months after the birth of the other; or whether this might not be a case, by no means uncommon, of twin-children—the one being born prematurely, and the other at the full period.

The consensus of medical opinion is that such a case is possible.

Admitting that both the children when born were mature, and therefore that it was a case of superfœtation, the first delivery must have taken place in the presence of witnesses; it would then have been known whether another child remained in the womb or not. If the two children were born within the common period of gestation after the absence or death of the husband, then their legitimacy would be presumed, until the fact of non-access was clearly established. The mere circumstance of their being apparently mature, and born at different periods, would *per se* furnish no evidence of their illegitimacy. On the other hand, if one or both of them were born out of the ordinary period, then, according to the evidence given, they might or might not be pronounced illegitimate. The law appears to have no sort of cognisance of the subject of superfœtation, as such: it appears to be merged in the question of protracted gestation.

Cases of different coloured children in the same pregnancy, by different coloured fathers, prove the possibility of superfecundation.

Maury[4] describes a case in which a well-formed fœtus 347 mm. long was found in the uterus and another fœtus, 11 mm. long, in the Fallopian tube. There were two corpora lutea at different stages of development, one in each ovary. Maury suggests that impregnation of two ova liberated at different times had taken place, for there was nothing to indicate that the smaller fœtus had been abnormally developed. Its age (six weeks approximately) corresponded with the corpus in the left ovary, and the age of the other (thirteen to fourteen weeks) corresponded with the age of the right corpus luteum.

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[2] Nurleos, B. C., *et alia*, *Brit. Med. Journ.*, 1: 1309 (1937).
[3] Smith, A. D. B., *Journ. Anat.*, 61: 319 (1927); 62: 100 (1927).
[4] *Jour. Surgery, Gynæc. and Obstet.*, vol. 40, May, 1925, p. 642.

The Average Period of Gestation

As a matter of fact, this is of little medico-legal interest, for in cases of dispute the question is not what is the average, but what is the possible duration of pregnancy. It is, however, well to state the generally accepted opinion, and to consider briefly the causes of difficulty in accepting a fixed period.

The usually accepted *average* is 280 days, which is the period allowed in ordinary clinical tables provided for medical use.

The difficulties in the way of exact determination are first and foremost the impossibility of fixing the exact moment of impregnation. Ovulation occurs in woman usually about 14 days before the onset of the ensuing menstrual period, but it is subject to many influences, and there are cases where it has seemingly occurred on almost any day in the intermenstrual cycle. It is generally accepted that the ovum has a very short life and probably does not survive more than 24 hours after ovulation unless it is fertilised. We have no means of knowing the exact moment of the union of the ovum and spermatozoon which is the beginning of pregnancy, but there is every reason to believe that the life of the effective sperm in the maternal passages is very short, probably less than thirty hours.[1]

It is generally supposed that parturition occurs at some anniversary of the menstrual period. Numerous facts tend to show that, notwithstanding the suppression of the menses, there is activity of the uterine system at what would have been, in the unimpregnated state, the menstrual period, and there is reason to believe that abortion takes place more readily at these than at other periods. Many observers consider that the duration of pregnancy is a multiple of the menstrual period; and that in the majority of women parturition will occur at what would have been the tenth menstrual period, or forty weeks from the date of intercourse and supposed conception. Labour usually starts about 280 days from the first day of the last menstrual period, so that the actual period of gestation is about 270 days or less.

There is no reason to believe that the *sex of a child* has any direct influence on the length of pregnancy. It has been stated that gestation is somewhat longer with male than with female children; and evidence of this kind was tendered in the Gardner Peerage case.

Watts Eden discusses the question of the duration of pregnancy at considerable length in the Transactions of the Medico-Legal Society, Vol. 17, and the related subject of ovulation is fully considered in the publications referred to below.[2,3,4,5]

To what Extent can Pregnancy be Shortened and yet a Viable Child be Born?

We here enter upon a subject which may be of the greatest importance, and upon which well-authenticated cases are practically the only means

[1] Moench, G. L., *Amer. Jour. Obstet. & Gynæc.*, 38: 163 (1939).
[2] Allen, *Journ. Amer. Med. Ass.*, 1930, p. 1080; Shaw, *B.M.J.*, January 6th, 1934.
[3] Dickinson, R. L., An ensemble on ovulation, *Journ. Contraception*, 3: 129 (1938).
[4] Hartman, C. G., *Time of ovulation in women*, Williams Wilkins & Co., Baltimore, 1936.
[5] Zeigler, S. L., *Fertility in Women*, Wm. Heinemann, London, 1945.

of deciding. Their evidence must be fully considered. Great mistakes have arisen in the calculation of the period by the use of the word "month" —some intending by this a *lunar* and others a *calendar* month. Nine lunar months would be equal to 252 days, while the average of nine calendar months would be 274 days, and would vary from 273 to 276 days—the period varying according to the particular months of the year over which the pregnancy might extend. To prevent mistakes, or that misunderstanding of evidence which has so frequently arisen, it would be advisable that medical witnesses should always express the period of gestation in weeks or days, concerning which there can be no misunderstanding: it would be proper also to adopt the plan of always commencing the calculation from the end of the last menstrual period, rather than from two weeks later. The latter rule is often followed, and this is another cause of confusion.

Premature Births. Short Periods of Gestation. We may regard all births before thirty-eight weeks as premature, and all those which occur after the fortieth week as protracted cases; and one great point for a medical witness to determine is, whether the characters presented by a child correspond to those which it should present if its period of gestation is of the alleged length. When the birth is premature, this sort of corroborative evidence may sometimes be obtained; because, assuming that there has been no access between the parties before marriage, children born at the fifth or sixth month after marriage cannot, if the offspring of the husband, present the characters of those born at the full period. It is not equally so with protracted births, for children are not necessarily more developed in protracted cases than they are in those which occur at the usual period. They may, however, and probably do grow during the whole period of intra-uterine life, for obstetricians have found that on an average children born after 280 days are heavier than those born before 280 days and are more apt to cause difficulties during parturition.

For an account of the features exhibited by children at different uterine ages, see "Age," Vol. I., page 125.

In judging from marks of development on the body of a child, we must make full allowance for the exceptions to which they are liable. The nearer the supposed premature delivery approaches to the full period of gestation, the more difficult will be the formation of an opinion; and if this opinion is to be of much scientific value it must be based on a series of X-ray photographs of the child to show the number, position and size of the centres of ossification of the bones. Thus, a child born at the eighth month may be the offspring of the husband—at the ninth month, of an adulterer; but medical facts could not enable a witness to draw any distinction. It is here that moral proofs are necessary, for without these the legitimacy of a child in such a case could not be successfully disputed. With respect to twin-children it is not unusual to observe considerable differences in age and height, especially when they are delivered from a single ovum. Cases have been recorded in which the respective weights of the twins were 4½ lb. and 2 lb., 5 lb. and 3 lb. 6 oz., 4 lb. and 1½ lb., and so on.

The fact that a child has had the strength to survive its birth for a certain period has been supposed to furnish additional evidence of

maturity; for it is well known that under a certain age children are not born living, or, if living, they speedily die.

Viability. According to English law, it is not necessary that a child, when born, should be capable of living, or *viable*, in order that it should acquire civil rights. Thus it may be born at an early period of gestation: it may be immature and not likely to survive; or it may be born at the full period of gestation, but there may be some malformation, defect, or disease, which must necessarily cause its death within a short time after its birth. Fortunately, these points are of no importance in relation to the right of inheritance: an English medical witness has to prove only that there was some physiological sign of *life* after birth (see "Live Birth"); whether the child were mature or immature, diseased or healthy, are matters which do not affect the question. In this respect English law appears to be more simple and just than that which prevails in France. By Art. 725 of the French Code, no child that is born alive can inherit unless it is born, as the law terms it, *viable*. The meaning of this word is not defined by the Code, and there are probably no two lawyers or physicians in that country who place upon it the same interpretation. There is no standard of viability provided the child in question is actually living at the time. *Solvitur ambulando* may well apply here.[1]

Earliest Period at which a Child may be born living. The question now to be considered in reference to English practice is, What is the *earliest period* at which a child can be born living, and with a capacity to live after its birth and attain maturity? Children born at the seventh month of gestation are capable of living, although they are more delicate, and in general require greater care and attention to preserve them, than children born at the ninth month. The chances are, however, very much against their surviving. It was the opinion of William Hunter, in which most modern authorities concur, that few children born before *seven calendar months* (or 210 days) are capable of living to manhood. They may be born alive at any period between the sixth and seventh months, or even, in some instances, earlier than the sixth; but this is rare, and, if born living, they usually die soon after birth.

Maisonneuve saw a woman two hours after delivery of a fœtus aged four and a half months; he then found the fœtus in its membranes, and on laying these open, to his surprise it was still moving. He applied warmth, and partially succeeded in restoring it, for in a few minutes the respiratory movements were performed with regularity, but in spite of the establishment of breathing the child died about six hours after its birth.[2]

Carter attended a woman who had an abortion when not more than *five months* advanced in pregnancy. The fœtus cried slightly directly it was born, and during the half-hour that it lived unsevered from its mother, it frequently tried to breathe. The body of the fœtus was one foot in length, and it weighed twenty and a half ounces. It appeared to be perfectly formed. From accurate information he was satisfied that the woman had not passed the fifth month of pregnancy.

In the following case a child born at the *fifth* month survived upwards of twelve hours:—

A woman in her second pregnancy and in the 147th day of gestation had severe flooding with rupture of the membranes. Labour occurred on the following night, when a small but well-formed fœtus was expelled giving no other indication of life

[1] Vibert, *Médecine Légale*, 513, 514.
[2] *Jour. de Méd.*; *Med. Gaz.*, vol. 39, p. 97.

than a feeble action of the heart, and a strong pulsation in the umbilical cord. It was resuscitated, and *cried* as strongly as a child born at the full period of pregnancy. It weighed less than two pounds, and was exactly twelve inches in length. It swallowed some nourishment, but died about twelve hours after birth. The pupillary membranes of the eyes were entire; the testicles had not descended; the head was well covered with hair. The length and weight, as well as the presence of hair, indicated a fœtus between the sixth and seventh months; but as it is asserted that the period of gestation is accurately given, this must be regarded as an extraordinary instance of premature development. There was clearly nothing in the organisation of this child to have prevented its growing to the age of maturity; in other words, it was *viable*.[1]

Routh reports a case in which a child born at five and a half months lived for eighteen days. The child was very small and weakly: its weight was not taken. The duration of pregnancy was twenty-two weeks and two days, or five lunar months and sixteen days. The child died on the eighteenth day after its birth.[2]

On a trial for child murder,[3] a midwife was indicted for causing the death of a child by bringing about the premature delivery of a woman when she was between the fifth and sixth months of pregnancy. The child in this instance lived five hours after its birth. Capuron mentions an instance in which a child was born after six and a half months of pregnancy: and at the time he reported the case, it was two years old and enjoyed excellent health. In another instance a child was born at he same period and lived to the age of ten years.[4]

Rüttel attended a married woman, who was afterwards delivered of a living child in the *fifth month* of her pregnancy: the child survived its birth for twenty-four hours. He delivered another woman of twins, in the *sixth month* of her pregnancy: one was dead, and the other continued alive for three hours, its life being indicated only by the visible pulsation of the heart—there was no perceptible respiration. This fact corroborates the remarks made elsewhere as to life without active respiration (see "Infanticide"). In another instance of the birth of male twins, at the *sixth month*, each weighed three pounds. Rüttel saw them a year after their birth, and they were then two healthy, strong children.[5]

Barker met with a case in which a female child was born at the 158th day of gestation or twenty-two weeks and four days after intercourse. The size and weight of the child corresponded with the period at which it was born; it weighed one pound, and measured eleven inches in length. It had only rudimentary nails, and very little hair on the back of the head; the eyelids were closed, and remained closed until the second day: the nails were hardly visible; the skin was shrivelled. The child did not suck properly until after the lapse of a month, and did not walk until it was nineteen months old. When born it was wrapped up in a box, and placed before the fire. Three and a half years afterwards this child was in a thriving state and healthy, but small, weighing twenty-nine pounds and a half.[6] Annan reported a case in which a child was born between the end of the sixth and middle of the seventh months, and lived for a period of four months and eight days. It weighed a pound and a half when seven days old.[7] In a case which occurred to Outrepont,[8] there was the strongest reason to believe that gestation could not

[1] *Med. Chir. Rev.*, July, 1844, p. 266.
[2] "Obst. Trans.," 1872, p. 132.
[3] *R. v. West*, Nottingham Lent Ass., 1848.
[4] "Méd. Lég. des Accouch.," pp. 162, 208; see another case, *Med. Gaz.*, vol. 32, p. 623.
[5] Henke's *Zeitschr.*, 1844, 241.
[6] *Med. Times*, 1850, 2, pp. 249, 392.
[7] *Med. Times*, 1848, p. 304.
[8] Henke's *Zeitschr.*, vol. 6.

have exceeded twenty-seven weeks. The child (a male) weighed, when born, one pound and a half, and was thirteen and a half inches in length. The skin was covered with down and much wrinkled; the limbs were small; the nails appeared like white folds of skin, and the testicles had not descended. It breathed as soon as it was born, and by great care its life was preserved.

It is singular that its development was very slow until it had reached a period which would have corresponded to the forty-second week of gestation. Outrepont saw the child when it had attained the age of eleven years, and it then appeared to be the size of a boy of eight years. The only remarkable point about the case is the length of time which the child lived. In one case,[1] a child born at six months and ten days was thriving satisfactorily when four months old.[2]

For a more recent case in which the period of gestation was 174 days see *Clark* v. *Clark*.[3]

In America a patient was delivered 192 days after the first day of her last period. The baby weighed two pounds eleven ounces (1.2 kilog.) and measured 35 cm. in length. It appeared premature and weak. At the time of the report it was a sixteen-months old, strong, healthy boy.[4]

Hence it is established that children born at the seventh, and even at or about the sixth month, may be reared.

The following case shows how the point may be of importance. It is known as the Kinghorn case.[5]

In 1838 an investigation (*fama clamosa*) took place before one of the Presbyteries of Scotland, in reference to certain reports which had been circulated to the prejudice of a minister of the district. His marriage took place on March 3rd, 1835, and his wife gave birth to a female child on August 24th following—*i.e.*, 174 days, or nearly *six calendar months* after the marriage—and the child continued to live until March 20th, 1836. When born it was very weak, and, according to the evidence of the accoucheur, and others who saw it, was decidedly immature. The birth of a living child, however, together with the fact of its surviving for so long a period, led to the report that there must have been intercourse between the parties previous to marriage: it was contended that the period was too short for the child to have been begotten in wedlock. Hamilton and Thatcher considered the complaint made against the minister groundless. The case went through several appeals, and was not finally decided until May, 1839, when the libel was found *not proven*, and the minister was absolved from censure.

Many medical witnesses gave evidence on the occasion; the majority of them were strongly in favour of this having been a legitimate and premature birth.

Although not connected with the medical part of the case, it should be observed that the character of the parties was free from all suspicion, that no concealment had been practised by them, and that no preparation had been made for the early birth of the child. There were, it is true, *unusual marks of development* about the child, considering the early period of its birth; yet these were not sufficient, any more than the fact of its surviving, to induce the belief that it had been conceived out of wedlock. It would be in the highest degree unjust to impute illegitimacy to offspring, or a want of chastity to parents merely from the fact of a six-months child being born living and surviving its birth.[5] There are, indeed, no justifiable medical grounds for adopting such an opinion—

[1] *Lancet*, 1851, 2, p. 177.
[2] See also *Med. Times*, February 16th, 1850, p. 129.
[3] [1939] 2; All E.R., 59.
[4] *Amer. Jour. Obs. and Gynæcology*, December, 1925.
[5] See observations of the President of the Divorce Division of the High Court of Justice in *Clark* v. *Clark* (1939), 2 All E.R., 59, at p. 60.

a fact clearly brought out by the answer to a question put to the principal medical witness in favour of the alleged ante-nuptial conception. He admitted that he had himself seen the case of a six-months child who had survived for *several days*. He could not assign any reason why, if after such a period of gestation it is possible to prolong life for *days*, it should not be possible to extend it to *months*.

Great injury to parties may be done by speculative medical opinion reflecting on the chastity of the parties concerned in cases of this sort.

Evidence from the State of Development. The fact that a child born at full term is small, and resembles in size and weight a seven- or eight-months child, cannot be taken as proof of illegitimacy. It has been already stated that children born at the full period vary considerably in size and weight; yet, although small, there are commonly about them the appearances of *complete development*. This is especially apparent in the features. If there is a general want of development of the body, and if certain fœtal peculiarities remain—as, for example, the pupillary membrane, or if, in the male, the testes do not occupy the scrotum—these facts lead to a presumption that the child has not reached the full period. On the other hand, when a child is born with the full signs of maturity, at or under seven months from possible access of the husband, then there is a strong presumption that it is illegitimate. The most progressive stage of development is considered to be during the last two months of gestation—the changes which the fœtus undergoes are greater and more marked at this than at any other time. The general opinion is that an eight-months child is not with any certainty to be distinguished from one born at the ninth month. If the body of a child is large and fully developed, it would in a general way be considered to have been born at the full period of gestation, and any opinion which had led to the supposition that it was a seven-months child would be attributed to some mistake in the calculation. The important question arises whether a seven-months child has ever been born so developed as to be mistaken by an experienced person for one that was mature. We know of no recent case to support such a belief.

The following case, in reference to development (at seven months), is well calculated to show the characteristics of a seven-months child, and to corroborate the views adopted by physiologists as to the means of determining the period of uterine life which the fœtus may have reached:—

A woman was married on April 7th, 1846, and was delivered of a male child at 7 p.m. on October 19th following, the period of gestation being equal to 195 days, or twenty-eight weeks. The infant cried strongly, and lived until 9 o'clock the following morning; the skin was of a deep pink or rose colour, beautifully soft, and covered with a fine down. The pupillary membranes were absent, and the pupils were well formed; the nails were complete; the testicles had *not* descended into the scrotum; the length of the body was fifteen inches, and its weight two pounds eight ounces. Its height, and the non-descent of the testicles, suggested a uterine age of seven months.

In addition to the other characteristics attributed to children born at the seventh month (see "Infanticide"), it may be observed that children at this uterine age do not so readily take the breast as those which have reached the ninth month, and their power of sucking is much more feeble.

Several cases have occurred in the Divorce Court in which the ability of a medical man to form an opinion of uterine age from the appearance of a child at birth, between the *seventh* and *ninth* months, has been seriously called in question. The use of the X-rays to determine the presence and size of points of ossification should add to the value of medical evidence in these cases.

In *Stone* v. *Stone and Appleton* (1864) the evidence showed that the husband went to India in August, 1859, and that he returned to England in May, 1861, and joined his wife on May 18th. The wife was delivered of a full-grown child on January 2nd, 1862, and the delivery at this date was assumed to be conclusive proof of adultery on her part. She was attended by a medical man, who deposed that in his opinion the child was full grown, *i.e.*, a nine-months child. Another medical man who saw the child two or three days after its birth, also considered it to be full grown. In comparing the date of possible access of the husband with the date of birth, the period of gestation would be 229 days or seven weeks and two days short of the average period.

The medical question was—Could this be the child of the husband? On the part of the wife, it was alleged that the child was a seven-months child prematurely born, and more than usually developed for its age; and evidence was given to show that in her previous deliveries the children had been prematurely born. Obstetric experts were also called to prove that any medical opinion based on the maturity or immaturity of the child was of no value; that out of a number of cases an experienced physician would be able to say with tolerable certainty in the majority whether a child was a seven-, or eight-, or nine-months child, but that he might be mistaken in certain cases. They also said that illness, bodily weakness, and mental anxiety tended to produce premature delivery, and that a woman who had once been prematurely delivered had a tendency to premature delivery, if she afterwards became pregnant. The jury returned a verdict for the husband, finding that the wife had been guilty of adultery; therefore that this was not the child of the husband, *i.e.*, it was not a seven-months child.

In another part of this work ("Uterine Age—Infanticide") some cases are related which prove that at the ninth month children are occasionally born of a size and weight exceeding the average. Thus an *alleged* nine-months child was born weighing eighteen pounds and measuring thirty-two inches, whereas the usual weight is from six to seven pounds and the length eighteen to twenty inches. In such an exceptional case there is reason to believe that had the child come into the world at the seventh month, it would then have appeared to the accoucheur to have reached the full term. It is impossible to say whether this case is one of post-maturity or not, though it certainly appears to be, but in any case in which this question arises, a witness will be bound to admit that great variations in size and weight occur, and he should not give a definite opinion about the age of a child without careful consideration of all the circumstances. In order to determine this point by unexceptionable facts, it would be necessary to collect a series of cases of impregnation from one intercourse in which the children were born seven months after such intercourse, and were proved to have had the average size and weight of mature children.

When the facts are such, that to be the offspring of the husband it must be a *six-months* child, and it is born *mature*, there can be no reasonable ground to doubt its illegitimacy.

Prolongation of Gestation

This question is even more important than that of shortened gestation. There must be unimpeachable evidence of the last possible chance of insemination. A rigid comparison can be instituted between periods of gestation only when there is definite evidence of the last possible chance of insemination; unfortunately, in most or many of the cases that follow, this method of estimation has not been followed, and allowance must be made; for all that, the cases are valuable and must be treated in full. Those which are the result of one coitus are naturally the most reliable.

That gestation may be retarded or protracted beyond the fortieth week is now, probably, not disputed by any obstetric writer of reputation. It is only by the accumulation of well-ascertained facts from all authentic sources that medical knowledge can be made available for the purposes of the law; otherwise, owing to the fact that a witness has not met with any exceptional instance, a court may be misled in its judgment by trusting to his opinion. It is the more important to attend to this, because most of the cases involving questions either of contested legitimacy, or the chastity of females, turn upon protracted rather than upon premature delivery.

Cases of protracted gestation are always open to the objection, either that the **menstrual function may have been suspended** from some hidden morbid cause, one or two months **before the actual date** of conception, or that there may have been some error in the calculation by which the period has been determined. If, however, the objection be admitted in these circumstances, it would be equally just to admit that in any given case the ordinary and so-called fixed period, calculated from the cessation of menstruation, is based on a fallacy. The menstrual function may have accidentally ceased, or continued for several intervals after conception, and thus a corresponding change should be made in fixing the ordinary period of gestation. This view of the question implies that no reliance can be placed on the date of the cessation of the menses as evidence of the actual duration of pregnancy, whether natural, premature, or protracted. Hicks met with a case in which the pregnancy of a woman appeared to be protracted to between twelve and thirteen months. There was every reason to believe that this woman had become pregnant during the absence of the menses, their suspension having taken place some time before intercourse; and this, no doubt, is the explanation of a large number of cases of alleged protracted gestation.

The cessation of the menstrual discharge must be either taken or rejected altogether as evidence: if taken, we have no right, in alleged protracted cases, to refer the suppression to disease, for the sake of shortening the period, when in ordinary cases we do not refer its continuance to disease, because this would tend to lengthen it; if rejected, it would be in the highest degree unjust not to give to a claimant the beneficial presumption of his having been born legitimate, when the cases adduced in evidence against his claim are actually based upon a precisely similar mode of calculation.

It is improbable that all the protracted cases recorded by observers are the result of mistakes made in the calculation of the period, since this calculation is based upon the same principles as those adopted in cases

of ordinary pregnancy. Hence, if there is a mistake in the one case, there would be in the other; if an error in the exception, there would be an error in the rule. Either the average term of pregnancy is wrongly calculated by most accoucheurs, at the thirty-eighth or fortieth week, or it is rightly calculated to extend occasionally to the forty-fourth or, admitting these protracted cases, to the *forty-sixth* week. But, even setting aside the obvious answer to an objection of this nature, some of the cases observed were instances of impregnation from a single intercourse; and, making due allowance for the interval for conception, the general inference would not be affected, and no fallacy would have arisen in such cases of protraction from mistakes dependent on the cessation of menstruation.

The real difficulty is that the cause of the onset of labour is not known. Many theories have been advanced, but even with the recent great progress in the knowledge of the hormonal control of the uterus, it is not yet possible to state the cause of labour with certainty. It is equally impossible, therefore, to be dogmatic as to the limit of time beyond which labour *must* necessarily terminate a pregnancy. No doubt there is a limit to gestation, but it is not in our power to fix it.

There is, indeed, only one point on which all modern observers agree, namely, that the period cannot be limited to a certain number of days, but that it is liable to variation according to circumstances but little understood.

It has been already observed that the date of intercourse does not furnish us with the date of conception, and according to some authorities all evidence connected with the function of menstruation is untrustworthy. In spite of these objections, the menstrual period must generally serve as a guide in default of more certain criteria.

Enge follows up the researches of von Winckel, and has made use of the register of the Leipzig Maternity for the seventeen years preceding the issue of this thesis.

In 175,333 births there were 821 children over 4 kilog. ($9\frac{7}{16}$ lb.) in weight and over 50 cm. ($19\frac{1}{2}$ in.) in length. The ratio of female to male infants was 100 : 110; 264 mothers had correctly reckoned term from the first day of the last period, 61 from the last coitus, and 81 from both factors. In 40 of these accurately reckoned cases the period lasted over 302 days, the maximum being 321. The average length of the children in these cases was 53-5 cm. (20 in.).

Such is a fair presentation of the arguments as based upon the opinions of those who were best qualified to judge. We may now give some reported cases and evidence which is strong enough in medical practice, and, *provided that other evidence is satisfactory*, one would think should be strong enough in law; but in *Robinson* v. *Robinson and Asplin* (a case communicated to Stevenson) **the judge refused to accept cases recorded in journals, etc., as evidence.** It is true, on the one hand, that the woman's statements in such cases are not sworn: but, on the other hand, they are usually given without any cause or motive for *mala fides*.

In *Gaskill* v. *Gaskill*[1] the court refused to grant a divorce where the husband, who was a soldier, had returned to duty 331 days before the birth of a large child, estimated to weigh about ten pounds. There

[1] [1921] p. 425.

was no direct evidence of adultery, but expert medical witnesses before the court gave evidence that there could be *no possibility* that the husband could be the father. In the opinion of the Lord Chancellor who heard the petition, however, the parties had been the sport of nature.

In works on midwifery will be found reports of cases in which gestation continued to the forty-first, forty-second, forty-third, and even to the forty-fourth week. Merriman published a Table on the subject of protracted gestation, on which the most experienced accoucheurs have been in the habit of relying. Of 114 pregnancies, calculated by him from the last day on which the women menstruated, and in which the children appeared to be mature, the following were the periods :—

In the 37th week . . .	3	In the 41st week . . .	22
„ 38th „ . . .	13	„ 42nd „ . . .	15
„ 39th „ . . .	14	„ 43rd „ . . .	10
„ 40th „ . . .	33	„ 44th „ . . .	4

In another well-marked case, birth occurred forty-four weeks precisely after the cessation of the menses.

From these results Merriman considered that in the greater number of women gestation is completed in the fortieth week from the cessation of the menses, and next to this period is the forty-first. The case of longest protraction on which he was able to rely was that of a married woman, who was in the habit of calculating from the last day on which her monthly period ceased. The lady was delivered 309 days, or forty-four weeks and one day, from the time at which she supposed that she had conceived. In another case the period was 303 days, or forty-three weeks and two days from the termination of the last monthly period.

A healthy woman, *æt.* 30, had borne three children, the youngest being four years old. She had menstruated regularly up to the third week in June ; the menses then stopped without any apparent cause. Her delivery took place 323 days after their last appearance. Allowing that impregnation occurred at the intermenstrual period, this would make the gestation 309 days ; or assuming that impregnation did not occur until twenty-eight days from the date of the last menstruation, this would make the period 295 days, or forty-two weeks and one day. Murphy furnished some facts in reference to this subject. Out of 182 cases, in which special inquiries were made of the women, the deliveries took place from the date of the last appearance of the menses at the following periods in weeks.

In the 33rd week . . .	5	In the 40th week . . .	25
„ 34th „ . . .	3	„ 41st „ . . .	32
„ 36th „ . . .	6	„ 42nd „ . . .	25
„ 37th „ . . .	11	„ 43rd „ . . .	19
„ 38th „ . . .	12	„ 44th „ . . .	9
„ 39th „ (9 months) .	24	„ 45th „ . . .	11

The most protracted of the cases in his Table was No. 182, where the period of gestation was 329 days, or, deducting twenty-eight days (the ascertained menstrual interval), 301 days, or forty-three weeks—*i.e.*, three weeks beyond the usual period.

All women may not have such unusually protracted pregnancies—indeed, it is well ascertained that no two women are alike in this respect, and that two successive pregnancies in the same woman are rarely alike in duration. Then, again, some medical men may not have met with protracted cases ; but the fact being clearly ascertained must be accepted, unless we doubt the credibility of reporters, well qualified to observe and having no conceivable motive to misrepresent the medical facts which came before them. The advocates of a fixed and a limitable period differ from each other by a space of at least ten or twelve days, and each must either take his own experience for the final decision of this question, or it must be allowed that men of equal powers of observation and experience with themselves have met with cases which have gone beyond their own fluctuating limits.

The question of how long a woman may carry an extra-uterine gestation that has mummified or become a lithopædion has no practical bearing on our present subject, with the possible exception that an extra- and an intra-uterine pregnancy occasionally have been known to occur simultaneously. The former might start a period of pregnancy, and the latter keep it up so as apparently to prolong gestation.

Analogy with other mammalia is admittedly a dangerous argument, but there is this to be said about the following Table, that the actual facts themselves in regard to coitus are beyond suspicion. A great variation in duration of gestation in the particular animals is therefore conclusively demonstrated.

Animal	Average Period	Days between Extremes	Percentage
Horses . . .	335	129	40
Cows	285	81	28
Sheep	153	11	7
Rabbits . . .	30	8	26

If we take the average of women as 280 days and allow them 80 days between extremes, this would mean 240 to 320 days as the limits calculated on the above Table.

The period of gestation with reference to legitimacy is further referred to on p. 62 *et seq.*

CHAPTER IV

DELIVERY

Delivery is a subject which much more frequently requires medico-legal intervention than pregnancy. In undertaking the investigation, we ought, if possible, to ascertain, either from the woman herself, or from those around her, whether there was reason to suspect that she had been pregnant. If we can obtain any information on this point, it may materially facilitate the inquiry. In the cases in which the law asks for proofs of delivery it often happens that pregnancy has been so concealed that few who saw the woman suspected her condition ; and, as the admission of her delivery may be the strongest proof of her criminality, she may resolutely deny it and a medical practitioner has no right to extort this admission from her. From this it will be seen that a medical witness must often be prepared to prove the fact of delivery against a woman who is criminally charged.

The uterus of a woman that has become pregnant can be emptied only in one of two ways, either naturally or artificially. The former constitutes delivery which may be premature or at full term ; the latter constitutes what the law defines as miscarriage ; in medicine it is convenient for purposes of description to distinguish between an abortion in the first three months, a miscarriage in the second three months, and a premature delivery in the third three months : the law makes no such distinction, but makes it a criminal act, no matter by what person, means or purpose effected (*vide* " Abortion ").

Cases not infrequently arise both in civil and criminal law in which this question of delivery, recent or remote, natural or artificial, becomes the all-important question to be decided by the medical witness. We shall consider it in the following order :—

Legal reasons for requiring proof of delivery.
Signs of recent delivery in the living.
,, ,, ,, ,, dead.
,, remote ,, ,, living.
,, ,, ,, ,, dead.

Legal Reasons for requiring Proof of Delivery

These may be enumerated as follows :—

(*a*) In connection with **legitimacy** or **suppositious children.**

(*b*) In **abortion** or **infanticide.**

(*c*) In **libel** actions or actions for **defamation of character.** These cases as a group do not belong to *medical* jurisprudence, and are therefore not further considered.

(*d*) For purposes of dealing with attempted " blackmail " or avoiding an attempted **compulsory marriage.**

(*e*) **In concealment of birth.**

In all of these the establishment of the *fact* of delivery may become of the very highest degree of importance for the purposes of justice, as is duly noted in the respective sub-sections, but it is only in the last case that the criminal law has a special clause referring to delivery *per se*.

Concealment of birth.[1] The Offences against the Person Act, 1861, s. 60, is as follows :—

If any woman shall be delivered of a child, every person who shall, by any secret disposition of the dead body of the said child, whether such child died before, at, or after its birth, endeavour to conceal the birth thereof, shall be guilty of a misdemeanour, and being convicted thereof, shall be liable, at the discretion of the court, to be imprisoned for any term not exceeding two years, with or without hard labour ; provided that if any person tried for the murder of any child shall be acquitted thereof it shall be lawful for the jury by whose verdict such person shall be acquitted to find, in case it shall so appear in evidence, that the child had recently been born, and that such person did, by some secret disposition of the dead body of such child, endeavour to conceal the birth thereof, and thereupon the court may pass such sentence as if such person had been convicted upon an indictment for the concealment of the birth.

This concealment of birth is an offence of which women charged with child-murder have been hitherto commonly convicted in England. English juries are very reluctant to convict women, in such circumstances, of the capital offence of murder, and consequently they often take advantage of the proviso to the effect that any person tried for the murder of any child, and acquitted thereof, may be found guilty of concealment of birth, if it shall appear in evidence that the child had recently been born, and that such person did by some *secret disposition of the dead body* endeavour to conceal the birth.

The medical evidence is derived as a rule from an examination of the mother ; much, therefore, will depend upon the time at which this is made. The body of the child need not even be produced, provided there be satisfactory evidence of its death. The body may have been secretly buried or burnt, and in the latter case it may be necessary to examine the ashes (see Vol. I). A medical witness may be required to give evidence of the age of the child or of the fœtus.

(By the Infanticide Act, 1938 (which does not extend to Scotland or to Northern Ireland) a child shall be deemed to have been recently born if it had been born within twelve months before its death.)

Proof of and what constitutes concealment are matters of law, and various interpretations have been placed upon the terms " concealment " or " secret disposition " of the body. This part of the evidence does not affect a medical witness, unless he himself has found the dead body or was present when it was found. It will rest with the judge to determine whether the body has been so disposed of as legally to constitute a misdemeanour. The law is especially lenient in such circumstances. A

[1] See also " Infanticide," *infra*.

very strict interpretation appears to be put upon this term "concealment." There must be a "secret disposition" of the dead body. A woman in London charged with "concealment" was acquitted because the evidence showed that the body of the infant was found on a rising ground in a field which was visible from a public highway. This was held not to be concealment.

In 1909 there was a case in which the body was placed in a box under the bed; this was not deemed concealment because the box was not locked.

It is not usual to find a married woman charged with this offence, but a man and his wife have been convicted of concealing the birth of a child.

The woman was delivered of a stillborn child early in the morning, and the husband buried the body in his garden, where it was afterwards found. Before the birth of the child the woman denied that she was pregnant, and after her delivery declared that she had not been aware of her pregnancy. The difficulty in the case was that no reasonable motive could be assigned for a husband and wife concealing the body of a dead child.

In a case under the Scottish statute of *concealment of pregnancy*, the following question arose, *viz.*:—"Whether the charge was excluded if the woman, an unmarried female, proved that she had intimated that she was with child to the father, but denied the pregnancy to everyone else. That the object of the statute was defeated in such a case, and yet that the main fact on which the statutory offence is founded was proved, could not be doubted. Concealment, and not calling and making use of assistance in the birth, constitute the offence. The Court of Justiciary was nearly equally divided. The majority went on the bare terms of the statute: the minority held that concealment was here a general term to denote the denial to all near and around the woman, and from whom assistance might be obtained, and was coupled with not calling for assistance in the birth."

Signs of Recent Delivery in the Living

These may be divided into general and local, and the two factors which govern them are the time that has elapsed since delivery, and the stage of growth and development which the expelled contents of the uterus had reached.

The signs which most frequently occur are as follows:—

Languid Look with Pulse and Temperature Slightly Increased. These are common in most slight illnesses, and are often found with menstruation. They disappear normally two or three days after delivery.

Peculiar Odour. Easily recognisable in the lying-in room of the poorer classes, and on turning down the bedclothes, in any woman. It is chiefly due to the vaginal discharge; it is perceived in many cases during menstruation. It disappears in a week or ten days with the discharge.

Breast Changes. These are full with a knotty feeling, and milk may be expressed; they are usually tender, and the areolæ are darkened. This condition of the breast is strong evidence, and lasts much longer than the other points. The pigmentation rarely disappears *entirely*, and hence is of some little value in estimating remote delivery. The drying up of the milk is extremely variable in its occurrence, and occasionally none appears at all, so that from the presence of milk it is impossible to

make any definite deductions as to *when* delivery took place. The presence of colostrum corpuscles in the milk, however, strongly suggests that parturition has taken place within a few days.

Abdomen Flaccid. A corroborative sign only, for the abdomen may be flaccid from many causes.

Lineæ Albicantes. These, whether recent or old, are simply evidence of previous prolonged distension of the abdominal walls or of the skin, for they are often seen in either sex when subcutaneous fat has been or is excessive; they are no proof that pregnancy was the cause of this distension, but, failing other possible causes, they are suggestive of past pregnancy. They are not always formed, or if formed they may disappear.

Uterus Enlarged and Easily Felt. This may have been caused by pregnancy, and may be suggestive, but there are other causes of uterine enlargement.

Perineum Lax and perhaps Torn. If the fourchette is intact, it is unlikely that the woman has had a full-time child: if it is ruptured, one may be certain that some large object has passed, or that some accident has happened to the vulva. A similar line of reasoning, though not quite so rigidly applied, may be used of the perineum—*i.e.*, the perineum does not rupture in parturition as easily as the fourchette: the greater the tear the more probably due to a full-time child; *per contra*, an intact perineum is no proof against a full-time child. The age of the tear in either case may be of value in fixing the date of delivery.

Vagina Lax and possibly Lacerated. This condition, if well marked, is valuable corroborative evidence, but (at any rate in multiparæ) the natural condition is too variable for the definite conclusions.

Os Uteri Flabby, Patulous, and perhaps Torn. This condition is strong evidence, for it is never found in a nulliparous uterus, except in the rare cases in which a polypus has been passed. This laxity, and the state of any lacerations of the cervix, are valuable indications as to the time of delivery. Blood oozing from the cervix is not a sign of much value, unless there is clear evidence that the woman has not suffered from a vaginal discharge. Endocervicitis is a frequent cause of such oozing.

The Lochia. These constitute strong evidence in the early stages (first three to four days). They usually cease to be bloody about the third day, and after then so much resemble other vaginal discharges as to be of little value as an indication of delivery.

The Zondek-Aschheim Test. If the blood or urine gives a positive test, it is strong corroborative evidence that pregnancy has recently terminated.

No one sign is conclusive, but the presence of many signs in combination affords very strong proof. They will vary in definition in proportion to the immaturity of the ovum, and in a case of delivery within two or three months of conception, there is very little prospect of distinguishing a miscarriage from menstruation, at any rate after twenty-four hours have passed. If the ovum, fœtus, or any of its membranes be found, then the fact of abortion may be proved. For the size of the embryo and fœtus at various ages, *vide* Vol. I, under "Age".

If any material delay has taken place before the examination is made, the signs which have been mentioned will have considerably diminished in definition: so that after a period, which is short in proportion to the earliness after conception, no traces whatever will be discovered.

A case is reported in which abortion took place, with a considerable loss of blood, at the end of the second month. Twenty-four hours afterwards, the mouth and neck of the uterus were almost completely restored to their natural state. The vaginal and external parts were hardly, if at all, dilated, and very little relaxed; the breasts exhibited imperfectly the appearances which accompany pregnancy, the ordinary symptoms of which had been almost entirely absent.

In such a case as this—and for such cases a medical witness must be prepared—scarcely a presumption could have been entertained of the fact of delivery. After twenty-four or thirty-six hours, in the greater number of these early cases, we may expect to find, from a personal examination of the woman, no proofs whatever of abortion.

In the later stages of pregnancy, the sum total of a combination of the signs is usually more conclusive, and the signs last longer, but even here delay is dangerous.

In some strong and vigorous women the body resumes its natural state within a few days, and the traces of parturition may have become so ambiguous as to furnish no satisfactory evidence. In others proofs of delivery may be obtainable for a fortnight or three weeks afterwards. In most cases, however, it is difficult to say, after the lapse of *eight or ten days*, that delivery has certainly taken place, the signs having partially disappeared. In all cases the earlier the period at which an examination is made, the more satisfactory will be the evidence obtained. Montgomery once examined a woman, *five days* after delivery at the full time and he was particularly struck with the degree to which the parts had been restored to their ordinary condition, especially the mouth and neck of the uterus, which hardly differed from their natural and unimpregnated form.[1] This inquiry becomes of considerable importance in a case of alleged infanticide. When the body of a child is not found until after two or three weeks from the time of its birth, and the suspected woman denies that she has been delivered of a child, she will probably not deny her pregnancy, but may assert that she has had an abortion at an early period. In cases of abortion at an early period the placenta is not always discharged at the time. A microscopical examination of the discharges might reveal structures of the placenta or chorion.

Medical men should refrain from giving an opinion based only on general symptoms: a woman who wishes to keep her confinement secret will make the most extraordinary efforts to maintain her usual habits, and, even without this inducement, a woman of the poorer classes will often resume her occupation almost immediately.

Tidy [2] saw a country woman doing heavy field work the day after the birth of her ninth child, and quotes the following extraordinary case:—

A girl, *æt.* 18, was delivered of a child during the night. The delivery caused so little disturbance as not even to excite the suspicion of any member of the family. The girl came down to breakfast as though nothing had happened, walked to the school where she taught, a distance of half a mile, and when her duties were over, returned in the evening. The next day she walked twelve miles, and was married on the fifth day after her confinement.

[1] "Cyc. Pr. Med.," *loc. cit.*
[2] "For. Med.," p. 128.

Parts of, or a whole fœtus may be found which incontestably prove that some woman has given them birth. The same may be said of the so-called vesicular mole or vesicular degeneration of the chorion, which is proof of conception, for it arises in no other way. When, however, nothing but a mass resembling a blood-clot is found, this must not be assumed to be a product of conception without a microscopical examination to show placental or fœtal tissues, for clots may be extruded in simple menstruation. The reader is referred to works on obstetrics for the microscopical appearances.

Signs of Recent Delivery in the Dead.

Here again it is possible that all the local signs mentioned above may be present, though the general signs will have disappeared ; the local signs will also be detected more easily, for the breasts may be cut open to look for milk, and may be examined under the microscope to show physiological activity.

The Excised Uterus. This will show more distinctly the laceration and bruising of the cervix. As regards its size this, of course, will vary with the period of gestation and the time after delivery at which death occurred ; it is flabby for a day or two and gradually resumes its firmness. The inner surface is for a day or two still bloody, and ragged looking, especially at the site of the placenta. The orbicular direction of the fibres around the internal orifices of the Fallopian tubes is at this time very distinct. In about a month the uterus will have become fully contracted ; but the mouth rarely closes so completely as in the virgin state. In a case in which a primipara, *æt.* 26, died from puerperal fever on the *sixth day* after delivery, the following appearances were met with in the uterus.

The internal surface was blackened and congested, especially in that part to which the placenta had been attached. There was here the appearance of suppurative action. The substance of the uterus was healthy ; there was no pus in the sinuses. The os uteri showed considerable ecchymosis. The vagina was healthy ; the iliac veins contained nothing but *post-mortem* clot.

An ecchymosed condition of the neck of the womb is very commonly found as the result of even an easy labour, and therefore forms a good guide when present. This point must be borne in mind in reference to criminal abortion, inasmuch as the neck has the appearance as if violence had been employed. From the statement of appearances given above, it will be seen that there must be considerable difficulty in determining the period prior to death at which delivery took place. The difficulty is increased when a woman has been prematurely delivered, or when death has not taken place until some time after delivery. An opinion may be in some degree strengthened by searching for those signs which have been described as characteristic of delivery in the living. These, if present, will always furnish strong corroborative evidence, not only of the fact of delivery, but of the period at which it had probably occurred. The absolute size is not of much value, for involution proceeds at a different rate in different women. A very similar condition of congestion of the interior is found after menstruation. The reader is referred to works on obstetrics for further details of the appearances in the two conditions.

The Placental Site. This affords proof of a recent pregnancy ; it is usually recognisable up to eight or nine weeks after delivery, and has

been noticeable as long as twelve weeks after. It is of very dark colour, looking sloughy and gangrenous, and the sinuses are very evident in it.

The Presence of a Corpus Luteum. Owing to the increase in our knowledge of the physiology of reproduction in the past few years the corpus luteum has been extensively investigated and its relationship to ovulation, menstruation and the maintenance of pregnancy discussed in great detail. Readers are referred to modern works on this special subject for details. It may be accepted that a corpus luteum is formed after each ovulation and that the secretion of the corpus is essential for the development of the uterine mucous membrane to prepare it for the attachment of the fertilised ovum. Should fertilisation not take place the corpus rapidly retrogresses, but in the event of pregnancy it increases in size and remains prominent for several months. There is, however, apart from its size and duration, no essential difference that we know of at present, between the corpus luteum of menstruation and that of pregnancy.

Investigations relative to pregnancy and delivery in the dead body are almost exclusively confined to cases of criminal abortion, where the contents of the uterus have been expelled at the sacrifice of the life of the woman. Death commonly ensues in these cases within two or three days after delivery and then satisfactory proofs are obtainable by a *post-mortem* examination, but if the woman has survived three or four weeks, it may be as difficult to determine delivery in the dead as in the living subject, except for the placental site. This remark applies to delivery at the full period ; for if the uterus has expelled its contents in the first months of pregnancy, the traces of this expulsion will have generally disappeared in the course of a few days.

Signs of Remote Delivery in the Living

A question may arise whether it is in the power of a medical practitioner to determine the period at which delivery took place, *i.e.*, how long a time has elapsed. This becomes necessary when, in cases of concealed birth, abortion, or infanticide (some time after suspected parturition), a child is found, and a medical witness is required to state whether the time which has elapsed since the birth of the child, either dead or living, corresponds with the supposed delivery of a suspected woman. An opinion may be possible if the examination takes place within eight or ten days after delivery, but it becomes difficult after the sixth day ; and when the tenth or twelfth day has passed it is still more difficult. After two or three months it may be regarded as impossible to assign the period of delivery with any degree of precision.

In a case of pretended delivery, contested legitimacy, or disputed chastity, a medical witness may be required to say whether a woman has, at any antecedent period of her life, been delivered of a child. Delivery has often been feigned by women for the purpose of extorting charity, compelling marriage, or disinheriting persons who have claims to an estate, and in other cases without any assignable motive. It may be said at once that unless the child, of which the woman is said to have been delivered, had been of at least six months' development, it is impossible to swear to the fact of her ever having been delivered at all, if we define " remote " as at least six months previous to examination. If it had reached this period some of the following signs may be found, but no one of them alone is of much use ; it is absolutely imperative for

many of them to be found, and then for other causes to be excluded which might account for them. They are :—

(*a*) **Breasts, Changes in.** There is commonly a so-called secondary areola on the breasts of parous women, and that rather darkly pigmented, but these points, especially the pigmentation, may fade after lactation, and may be found in non-parous brunettes ; the presence of the secondary areola is a little more reliable. The breasts in parous women are commonly pendulous, but the breasts of any woman may become pendulous from many other causes than a past pregnancy, and they may recover their shape and firmness very well after a pregnancy, and even after prolonged lactation. If lineæ albicantes (the little white, atrophic, glistening lines) can be found they are so far proof that the skin over the breast has been at some time greatly distended, but do not prove that pregnancy was the cause of it ; excessive fat, or even an abscess, might cause such distension.

(*b*) **Abdomen with Lineæ Atrophicæ.** The same arguments apply as to those in the breast, only that the possible causes are even more numerous than for the breast – ascites, tumour, œdema, fat, etc.

(*c*) **Vulva and Perineum.** It is much easier, from the completeness of the posterior commissure, or absence of any rupture of perineum, or of a scar of such rupture, to say that probably there has not been parturition, than it is to state positively that there has been parturition because the appropriate opposite to the above is present ; disease and accident may easily be alleged as the cause of what is found.

(*d*) **Hymen.** An unruptured hymen is, of course, absolute proof against parturition having ever occurred, although it is no proof of virginity ; if the attached margins of the fragments of lacerated hymen are in continuity with one another it may safely be sworn that no full-time child has passed ; if *caruncul e myrtiformes* (these are the tags of tissue left by the ruptured hymen which do not touch at their seat of attachment) are present, this is conclusive proof of considerable distension of the hymeneal orifice, but things other than a fœtus may have done this.

(*e*) **Uterus Enlarged, and Cervix Lacerated.** It is true that when once the uterus has carried a fœtus for many months it never again becomes quite so small or quite the same shape as one which has not done so ; the differences are, however, such that in the living (even healthy) uterus they could not be positively asserted to be due to a past pregnancy, while disease such as fibroids will quite destroy any chance of *ante-mortem* distinction between a parous and non-parous uterus. The condition of the cervix is more reliable (*vide* above "Signs of Recent Delivery"), though, of course, such lacerations may have entirely disappeared. The external os in the nulliparous uterus is usually a small circular dimple, while after parturition it is commonly seen as a longitudinal slit.

Signs of Remote Delivery in the Dead

Death will not have destroyed any of the signs mentioned as applicable to the living unless decomposition or some other destructive agency has been at work too long. The uterus may be more closely investigated and measured, the shape and size of its cavity and cervix measured, and also any disease such as fibroids can be for a certainty detected. Microscopic sections of the uterine wall will show numerous vessels obliterated by endarteritis in the parous organ. For elaborate differences between the parous and non-parous uterus *vide* works on obstetrics.

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CHAPTER V

DISSOLUTION AND NULLITY OF MARRIAGE

Legal Aspects of the Subject

In the contract of marriage, capacity of consummation is implicit, so that an incapacity in either party in this respect may constitute a legal ground for annulling the marriage.

Dissolution of Marriage. Since the operation of the Matrimonial Causes Act, 1937, a petition for dissolution of marriage may be presented by either spouse on the ground of adultery, cruelty, or desertion without cause, or incurable unsoundness of mind, or on the ground that the spouse can be presumed to be dead.

The wife may, in addition, present a petition on the ground that the husband has been guilty of rape, sodomy, or bestiality. No such petition may be presented within three years of the marriage, in the absence of proof of exceptional hardship suffered by the petitioner, or of exceptional depravity on the part of the respondent, and by leave of the Judge.[1]

A husband cannot obtain relief on the ground of unnatural practices by his wife.

Judicial Separation. A petition may be presented on any of the above-mentioned grounds applicable to divorce, or on the ground of failure to comply with a decree for restitution of conjugal rights, or on any other ground on which a decree for divorce *a mensa et thoro* might have been pronounced immediately before the commencement of the Matrimonial Causes Act, 1857.

Nullity of Marriage.

(A) Marriages void *ab initio* on the ground of bigamy, want of consent caused by duress or fraud, consanguinity, insanity of one of the parties at marriage, or irregularity as to forms and ceremonies.

(B) Marriages voidable only on the ground of impotency, malformation, frigidity, incapacity at the time of the marriage to consummate the same; wilful refusal to consummate the marriage, insanity or epilepsy, or mental deficiency at time of marriage, venereal disease in communicable form at time of marriage, wife at time of marriage pregnant by a man other than the petitioner.

The three last-mentioned grounds are subject to the court's being satisfied that the petitioner was at the time of the marriage ignorant of the facts alleged; that proceedings were instituted within a year of

[1] *Rayden on Divorce*, 4th Ed., pp. 35 *et seq.*

the marriage; and that marital intercourse with the consent of the petitioner has not taken place since the discovery by the petitioner of the existence of the grounds for a decree.

Summary Jurisdiction (Married Women and Separation and Maintenance) Acts, 1895 to 1925 (as extended by the Matrimonial Causes Act, 1937, s. 11). Under these statutes a court of summary jurisdiction may order that a wife whose husband has been convicted summarily of an aggravated assault upon her within the meaning of the Offences against the Person Act, 1861; or has been convicted on indictment of an assault upon her, and sentenced to pay a fine of more than 5*l*., or to a term of imprisonment exceeding two months; or has deserted her, or has been guilty of persistent cruelty to her or to her children, or of wilful neglect to provide reasonable maintenance for her or her infant children whom he is legally liable to maintain; or while himself suffering from a venereal disease, and knowing that he was so suffering, has insisted on having sexual intercourse with her; or has compelled her to submit herself to prostitution, or whose husband, not being amenable to any jurisdiction in lunacy, is, by reason of habitual drunkenness or the habitual taking of dangerous drugs, at times dangerous to himself and others or incapable of managing himself and his affairs, need no longer cohabit with her husband. A certificate of a conviction for cruelty may be used to corroborate a wife's allegations of cruelty in the Divorce Court[1]; but apparently the depositions taken before the justices may not be so used.[2] In addition, a wife is entitled to relief where the husband is found by the court to be an habitual drunkard under the provisions of the Inebriates Acts, 1879 to 1898, and the Licensing Act, 1902 (as amended by sect. 3 of the Summary Jurisdiction (Separation and Maintenance) Act, 1925). By the Matrimonial Causes Act, 1937, an order may also be made where the husband has been guilty of adultery, but an order cannot be made unless the court is satisfied that there has been no condonation or connivance, or conduct conducing, or collusion. The complaint in such cases must be lodged within six months of the commission of the offence (*Teall* v. *Teall*).[3]

Legal Cruelty. The definition of this matrimonial offence was laid down by Lord Stowell in 1790, in *Evans* v. *Evans*,[4] and was approved by a majority of the House of Lords in *Russell* v. *Russell*.[5] It is described as conduct of such a nature as to have caused **danger to life, limb, or bodily or mental health, or as to give rise to a reasonable apprehension of such danger.**

Either a husband or wife may be found guilty of this offence.[6] It is seldom that one act of cruelty is so serious as to be deemed sufficient by the court; but where there are continued acts of bad treatment and ill-usage they will be considered as cruelty when taken together.[7] Where the petitioner's health has broken down owing to a long course of conduct on the part of the other spouse, the court will give relief. Wilfully and recklessly to communicate syphilis or gonorrhœa is cruelty.[8]

[1] *Harriman* v. *Harriman*, [1909] P. 123.
[2] *Judd* v. *Judd*, [1907] P. 241.
[3] [1938] P. 250.
[4] Hag. Con. 35.
[5] [1897] A. C. 395.
[6] See *Atkinson* v. *Atkinson* (1925), *Times*, March 26th.
[7] *Hadden* v. *Hadden* (1919), *Times*, December 5th.
[8] *Browning* v. *Browning*, [1911] P. 161.

In *Garner* v. *Garner*[1] it was held that a physician can be compelled to give evidence of venereal disease, although he was treating the patient under a national scheme which enjoins absolute secrecy. It would appear that in the absence of other and more serious acts it is not cruelty to infect a husband or wife with a mere skin disease.

A man cannot be convicted criminally of unlawfully and maliciously inflicting grievous bodily harm on his wife, or of occasioning her actual bodily harm, by reason of his having communicated to her a venereal disease from which he was suffering, although at the time of his having connection with her he was aware, and she was unaware, of his condition, *viz.*, gonorrhœa.[2] Drunkenness of itself is not regarded by the court as cruelty, but a petitioner will be entitled to relief if the acts complained of were committed while under the influence and effects of drink.[3]

The court will protect a petitioner from cruelty by an insane spouse where the insanity is recurrent and intermittent, and the safety of the petitioner is in question.[4] But it would appear from the remarks of Lord Gorell in Baron's case that, if the insanity is of a permanent nature, the remedy of the petitioner would be to apply the provisions of the Lunacy and Mental Treatment Acts. (But see now the Matrimonial Causes Act, 1937.)

Threats of actual personal violence sometimes constitute legal cruelty; but mere vulgar abuse, obscene language, false accusations of adultery, or even the act of spitting in a wife's face will not, unless accompanied by other acts. Words alone, however galling, however violent, and even if they impute a crime of the most disgraceful kind, have been held not to constitute legal cruelty. Such conduct as the above, with other acts, such as false accusations of adultery[5] or unnatural practices, may be regarded as coming within the definition and the principle of cumulative cruelty.[6]

Sexual Malpractices apparently do not in themselves constitute cruelty without proof that the practice of them or invitations to repeat them have caused injury to health.[7]

Medical Aspects of the Question [8]

Dealing with these in order, medical evidence may be required to prove: **Adultery on the part of the wife.** This naturally involves the questions of prolonged and shortened gestation if she should have become pregnant as the result of such intercourse: this we have already considered in full. In ordinary circumstances, the case calls for no other medical evidence (except perhaps that of identity), and no further notice will be taken of it.

[1] (1920), 36 T. L. R. 196.
[2] *R.* v. *Clarence* (1888), 16 Cox, 511.
[3] *Walker* v. *Walker* (1898), 77 L. T. 715.
[4] *Hanbury* v. *Hanbury*, 1892 P. 222—husband suffered from intermittent and recurrent mania; *Baron* v. *Baron* (1908), 24 T. L. R. 273—where the husband had twice attempted suicide, necessitating treatment in a mental hospital for a few months, and on other occasions made threats against wife, so that her health became impaired.
[5] *Jeapes* v. *Jeapes* (1903), 89 L. T. 74.
[6] *Russell* v. *Russell*, [1897] A. C. 395.
[7] *Statham* v. *Statham*, [1929] P. 131.
[8] For a paper entitled "Some Medical Aspects of the Law of Nullity," by S. H. Belfrage, M.D., see *Trans. Med.-Leg. Soc.*, 1923–24

A husband cannot obtain a decree of nullity of marriage on the ground of his wife's fraud in concealing from him the fact that she was pregnant by another man at the date of the marriage.[1]

Communication of Venereal Disease. It is difficult in view of the legal aspects of this question to make any general statements on this subject, but gonorrhœa is frequently the subject of medico-legal inquiry from its widespread dissemination and the injury which may be produced in the female if the uterus, tubes, or ovaries become infected. The only accurate method of diagnosis is the bacteriological demonstration of the gonococcus. A certificate of freedom from gonorrhœa to a marriage candidate should be granted by a physician only after the greatest deliberation and after repeated negative examinations by a bacteriological expert. The absence of any discharge clinically is no evidence of the absence of the gonococcus even years after an attack of the disease, and as long as the organism is present the patient is capable of communicating the disease.

De Lisle v. *De Lisle*.[2] In this case the wife sought a divorce by reason of the adultery and cruelty of her husband, the cruelty consisting in the alleged transmission of gonorrhœa, whereby her health was seriously affected. The husband having had gonorrhœa within a year of marriage received a certificate from a medical practitioner that he was in good health on June 26th, 1902. On July 9th marriage took place, and was consummated in a violent manner, the wife being severely lacerated. Between July 10th and 12th connection took place four times, on each occasion accompanied by severe pain. On July 13th menstruation ensued and lasted some eight days, and marital relations were not resumed until July 27th, when at the request of the wife connection took place and continued twice daily until August 2nd. On all these occasions the wife was free from pain. On August 3rd connection was attempted, but failed, as the husband was unable to get an erection. On August 4th the husband complained of feeling unwell, and consulted a doctor, but did not inform his wife of the result of his medical consultation. On August 9th, *i.e.*, six days after last coitus, the wife noticed a discharge on her linen and felt smarting pains on micturition. Early in September she consulted a physician in Rome, as she had doubts whether her marriage had really been consummated. While answering this question in the affirmative, the physician in question was apparently led from the appearances presented to ask for an examination of the sheets of the bed occupied by the wife during her last period of marital intercourse. Stains were found on the sheets, and cocci similar in appearance to the gonococcus, and the wife was thereupon seen by a venereal specialist, who pronounced the disease to be clinically gonorrhœa, and on examination of the pus the gonococcus was stated to have been found. The examination was microscopic only. No cultures were made. On the night of October 7th violent uterine hæmorrhage ensued, with the result that on the 9th the uterus was cleared out, and a putrefied fœtus was found. The wife was ill for many weeks, and as the result of her sufferings brought the action against the husband. Medical witnesses were called for both parties, the defence being that the wife did not suffer from gonorrhœa, or if she did that it was an infection from a latent gonorrhœa in the husband, he believing himself to be cured before the marriage took place. For the wife the medical expert opinion was that although no cultivation of the gonococcus had been made, the whole history of the case, together with the microscopic diagnosis, was proof of the existence of gonorrhœa. In the cross-examination the question of the existence of pseudo-gonococci was considered, also the possibility of the cause of the condition being due to leucorrhœa. On the other hand, the medical experts for the wife laid great stress on the fact that had the gonorrhœa been contracted from a latent gonorrhœa in the husband, the discharge would have made its appearance long before August 9th, *i.e.*, thirty-four days after marriage. The fact of its appearance on this date was held by the

[1] *Moss* v. *Moss, otherwise Archer*, [1897] P. 263.
[2] (1904) *The Times*, March 15th.

medical experts to be conclusive evidence that there had been a fresh adulterous infection in the husband some time between July 13th and August 3rd, and probably in the latter part of this period.

For the defence it was maintained that the disease from which the wife suffered had never been a gonorrhœa, that it might have been set up by a leucorrhœa the result of the violent intercourse with laceration which took place in the first instance, and that a mere microscopic diagnosis was totally insufficient to diagnose the gonococcus, which might be closely simulated in appearance and staining reactions by other cocci in the urethra or cervix uteri. The husband in cross-examination denied adultery, but admitted having had three separate gonorrhœal infections before marriage. He also admitted having consulted doctors about his genitals before he was even aware that the wife had contracted a discharge.

In giving judgment, granting a decree nisi, the judge held that the diagnosis of gonorrhœa had been established, even although the final test, the cultivation of the gonococcus, had not been carried out. He dealt also with the inability of the defence to establish a diagnosis other than gonorrhœa. From the length of the incubation period it was held that the gonorrhœa must have been contracted by the husband subsequently to marriage, the inability to have connection on August 4th and the subsequent visit to the doctor being evidence that the disease was then commencing. To establish the charge of cruelty, the judge held that it must be shown that the husband had a knowledge that he was suffering from the disease, or at least a suspicion that he was suffering, and this was proved by the fact that on more than one occasion he had consulted medical men before he was aware that the wife had contracted it.

Nullity of Marriage for alleged impotency or sterility.[1] Impotency may arise either from malformation, or from that which the canon law describes as "frigidity of constitution," or from any physical cause of whatever nature which may render intercourse impossible. A petition for nullity may be presented by either party, and the medical proof required must be such as to satisfy the court that the incapacity alleged was in existence at the time of the marriage, and that it still remained without remedy.

In a case which came before the old Ecclesiastical Courts a singular question arose whether, when there was a capacity for sexual intercourse on the part of a woman, with a certainty that from physical defect it could never be prolific, this was sufficient to entitle the husband to a divorce.

The woman was examined by three physicians; and they reported that the sexual organs were undeveloped, like those of girls who had not reached puberty, that the vagina was only three-quarters of an inch in depth, and that there was no uterus. They stated that sexual intercourse might take place in an imperfect way, but that conception could never result. On a second examination, seven months afterwards, it was found that the vagina had become elongated, and had then a depth of two inches; but there were no medical means of improving its condition or of removing the defect. It was contended for the husband that the defect was natural and irremediable, and that he was entitled to a sentence of nullity of marriage. On the part of the wife, it was insisted that, in order to entitle a party to this sentence, there must be an *utter impossibility* of sexual intercourse. The case, it was argued, was one of mere sterility, which was no ground for a decree; actual consummation had taken place.

Dr. Lushington, in pronouncing the marriage null and void, said that mere incapability of *conception* was not a sufficient ground whereon to found a decree of nullity, and that the only question was whether a woman was or was not capable of sexual intercourse. In his opinion, a power of sexual intercourse was necessary to constitute the marriage bond:

[1] For papers on Nullity and Impotence by Dr. E.A. Bennett and Mr. Adrian Stephen, see *Medico-Legal and Criminological Review*, 1936, pp. 36 and 42.

and the intercourse must be ordinary and complete, not partial and imperfect ; yet it would not be proper to say that every degree of imperfection would deprive it of its natural character.

Negative evidence of non-consummation from the physical condition of a woman is of much less value, *cæteris paribus*, than the affirmative evidence from the existence of a physical defect in a man (*vide* "Impotence"). When the defect is not apparent on an examination, the case is attended with considerable difficulty. Dissolution of marriage has, however, been granted even in these cases, when the husband has acknowledged his incapacity and when, notwithstanding cohabitation for some years, this admission has been confirmed by an examination of the wife. Even when the male organs do not appear well developed, if sexual desire is absent, great caution is required in drawing up a report. In investigating a case of this kind, when there is no apparent physical defect or malformation, it is necessary to examine the bodily state of the person, whether he is effeminate, or, on the other hand, whether there are any or all of the usual marks which attend the virile state. In the latter case the impotency may be only temporary ; and it would be decidedly unsafe to pronounce an opinion adverse to the existence of procreative power.

In order to justify a declaration of nullity on the ground of impotency or sterility, the impediment to intercourse or procreation should be established by good medical evidence, and the condition must be *evident* and *irremediable*. It must also have existed before the marriage, and have been entirely unknown to the petitioner. The nature of the impediment should be determined by private medical opinions or affidavits, based on an examination of *both* parties. Although there is no reason whatever to suppose that impotency and sexual malformation are more common in males than malformation and sterility in females, we rarely hear of a husband instituting proceedings for nullity of marriage on the ground of sterility (incapacity of procreation) in the wife ; and in most instances the wife petitions on the ground of impotency or incapacity of intercourse in the husband. The difficulty of establishing incapacity in a woman, and the facility of proving impotency from physical causes in a man, may possibly account for this difference.

The malformation described in the following continental case is probably not infrequent among reputed females. If not detected at birth, it may be detected at the age of puberty, and the unfortunate consequences of a matrimonial alliance prevented.

The husband, *æt.* 29, alleged that his wife was incapacitated for sexual intercourse, and demanded a separation from her. An examination of the husband showed that there was no deficiency or defect on his part. The wife, *æt.* 26, was a healthy-looking person. Her voice was rough, like that of a man, with shrill tones resembling those of a boy at puberty. The pomum Adami (larynx) projected as in a man. There was no appearance of breasts ; the face was not hairy. There were some stiff hairs on the upper lip and chin. The pelvis was that of a male. There was a penis about one inch and a half long and one inch in diameter, with prepuce and glans, but no perforation for the passage of urine. This was below and behind the root of the organ (*hypospadias*, p. 17, *ante*). There were projecting labia, with a deep fissure between, in the situation of the vagina, about two inches in depth, and getting narrower as it proceeded backwards. No uterus could be felt. In one labium there was a perfectly well-formed testicle. The other contained none, but a testicle of smaller size was found in the inguinal canal.

The wife was well aware that she was not like other women. Her parents had concealed her condition from her, and had never consulted a medical man

She admitted she had never menstruated, and had not had any mucous discharges from the vagina. She had experienced but little sexual feeling, never towards men, but more towards women. She regretted her condition, and agreed to a separation from her husband. The wife was an hermaphrodite, with more of male than female development, she was incapacitated for sexual intercourse as a woman, the sexual defect was congenital, *i.e.*, existing before the marriage, and was incurable. The marriage was declared void, and the wife was ordered to put on the clothes of a man.

In an unreported petition for nullity of marriage, there was anchylosis of both hip-joints in the wife. Nevertheless there was abundant evidence that there had been repeated sexual intercourse from behind (*more ferarum*).

In the following unreported case the question arose whether nullity petitions were restricted to the husband and wife during life, or whether third parties could intervene to promote them for their own interest after the death of either.

The plaintiff claimed a right to administer the estate of his deceased wife who had died intestate. He made the claim as her lawful husband. The next-of-kin of the wife, who were the defendants in the case, contended that, by reason of physical incapacity, the marriage with the intestate was void, and he was not the lawful husband. He therefore had no legal right to claim administration.

Sir J. Wilde said—

A distinction must be made between void and voidable. In cases of physical incapacity the marriage is not void, but voidable under certain conditions. Thus the party complaining must be sincere on the ground on which he is asking relief, and the physical defect must be incurable. This matter of incapacity ought to be raised only by the person who suffers any injury from it, and who elects to make it a ground for asking that the contract of marriage should be annulled. Such a question can only be discussed and adjudicated in the lifetime of the parties. It is a matter of *personal* complaint only, and has always been dealt with as such. In this suit the rights of third parties had been introduced. The question whether two persons are married or not may arise on a variety of occasions, and be raised by third persons, as creditors or otherwise. Now if the parties themselves are content with the *consortium vitæ*, and prefer to maintain the bond of matrimony intact, would it not be almost intolerable that a third person should have a right to insist upon an inquiry into the nature of their cohabitation and the revelation of their physical defects? The ground of nullity must therefore be confined to a suit brought by one of the parties to the marriage in the Matrimonial Court.

The learned Judge gave judgment for the husband, whereby, notwithstanding physical incapacity, he was constituted administrator of his deceased wife's estate.

It would seem to be the duty of the court to determine in which of the parties the disability lay; but in *G.* v. *G.*[1] the court pronounced a decree *nisi* of nullity on both cross-petitions, and left the question undetermined.

In *C.* (*otherwise H.*) v. *C.*,[2] the House of Lords held that impotency is a ground for a decree of nullity, inasmuch as the husband was incapable of consummating that particular union with that particular woman.

In nullity suits on the ground of impotence, an order for the appointment of two medical inspectors must be obtained from a Registrar of the Divorce Division. After examination of the parties the inspectors

[1] [1912] P. 173 (followed in *H.* v. *H.* (*otherwise N.*) (1929), 45 T. L. R. 618).
[2] [1921] P. 399.

make a report to the Registrar. Both parties are entitled to a copy of the report. The refusal of a respondent in a nullity suit to be examined does not prevent the suit from proceeding. If a nullity suit be contested (which is rare), there may be other medical evidence in addition to that of the inspectors.

The following cases are taken from the latest (1942) edition of Rayden's " Practice and Law in the Divorce Division " :—

(*a*) **Nullity Decrees Granted.** *D.* v. *A.* (*otherwise D.*).[1] Where wife had no uterus, the vagina forming a *cul de sac*—complete *coitus* being impossible.

W. v. *H.* (*otherwise W.*).[2] Consummation impossible owing to congenital malformation of wife, which was removable only at considerable risk to her life.

G. (*otherwise T.*) v. *T.*[3] Parties separated at end of three months. Wife had imperfect signs of virginity or of connection, husband believed to be incurably impotent.

G. v. *G.*[4] A wife of middle age had successfully resisted husband for about three years—no malformation, but refused to submit to remedies owing to dangerous nature.

P. v. *L.* (*otherwise P.*).[5] Wife aged eighteen and hysterical. Struck husband when attempts were made. Threats to commit suicide. Said she had no sexual desire and refused remedies.

H. v. *P.* (*otherwise H.*).[6] After three years' cohabitation, husband's attempt excited hysteria. Wife refused examination.

L. v. *L.* (*otherwise W.*).[7] Wife aged twenty-three, with vaginismus, slept on occasions with husband for three years. Slightly dangerous operation refused.

F. v. *P.* (*otherwise F.*).[8] Respondent a widow, not a virgin, without children. Slept with second husband five months. Non-consummation admitted. Decree granted on ground of " latent incapacity arising from hysteria." [9]

G. v. *G.*[10] All attempts resisted by wife over protracted period.

B. (*otherwise H.*) v. *B.*[11] Eight months after the marriage separate beds against wish of wife. Subsequent attempts in same bed. Wife unwillingly executed deed of separation. Husband refused examination.

R. (*otherwise K.*) v. *R.*[12] Before marriage, wife seduced by another man. Husband admitted impotence.

S. v. *S.* (*otherwise M.*).[13] Wife refused. No attempt by husband. Examination refused.

[1] (1845), 1 Rob. Ecc. 279.
[2] (1861), 2 Sw. & Tr. 240.
[3] (1854), 1 Ecc. & Ad. 389.
[4] (1871), 2 P. & D. 287.
[5] (1873), 3 P. D. at foot of p. 73.
[6] (1873), 3 P. & D. 126.
[7] (1882), 7 P. D. 16.
[8] (1896), 75 L. T. 192.
[9] *Cp. V.* v. *V.*, otherwise *C.* (1921), 37 T. L. R. 322.
[10] [1924] A. C. 349.
[11] [1901], P. 39.
[12] (1907), 24 T. L. R. 65.
[13] (1908), 24 T. L. R. 253.

J. (*otherwise K.*) v. *J.*[1] Where husband refused to give up habit of self-abuse and left wife.

L. (*otherwise B.*) v. *B.*[2] Wife delayed bringing suit for seven years.

S. v. *B.* (*otherwise S.*).[3] Decree granted seventeen years after marriage.

(*b*) **Decrees Refused.** *U.* (*falsely called J.*) v. *J.*[4] Assertion of wife not accepted by court. Medical evidence uncertain. Denial by husband.

S. v. *A.* (*otherwise S.*).[5] Husband and wife had lived together nine years, but court was not satisfied as to husband's attempts.

S. (*falsely called E.*) v. *E.*[6] Permanent incapacity not assumed by court. Two attempts during three months' cohabitation, unsuccessful owing to husband's habit of self-abuse, probably curable. Wife's health affected.

Finegan v. *Finegan* (*otherwise McHardy*).[7] Pre-nuptial agreement that no intercourse until wife agreed ; no grounds for inferring incapacity.

Hudston v. *Hudston* (*otherwise Newbigging*).[8] Persistent refusal, but no ground for inferring incapacity ; pre-nuptial agreement for non-consummation alleged, but not proved.

[1] (1908), 24 T. L. R. 622.
[2] [1895] P. 274.
[3] (1905), 21 T. L. R. 219.
[4] (1867), 1 P. & D. 460.
[5] (1878), 3 P. D. 72.
[6] (1863), 3 Sw. & Tr. 240.
[7] (1917), 33 T. L. R. 173.
[8] (1922), 39 T. L. R. 108.

CHAPTER VI

LEGITIMACY AND PATERNITY:

Legal Presumption of Legitimacy

Every child born either in lawful matrimony, or within a period after the death of the husband in accordance with the natural duration of gestation, is considered by the English law to be the child of the husband, unless the contrary be made clearly to appear by medical or moral evidence, or by both combined. It is only in reference to *medical* evidence that the subject of legitimacy can here be considered; but it is extremely rare to find a case of this kind determined by medical evidence alone. As a rule, there are circumstances which show that a child whose legitimacy is disputed is the offspring of adultery, whereas the *medical* facts may be perfectly reconcilable with the supposition that the claimant is the child of the husband. Very often, therefore, these cases have been decided from *moral* evidence alone, because the medical evidence of the period of gestation or of physical capacity in the parties left the matter in doubt. The common law of England was, according to Lord Coke, to the effect that if a child was born during marriage, the husband being within the four seas of the realm (*intra quatuor maria*), and no physical impossibility being proved, the child was legitimate. Access was presumed, unless it could be proved that the husband was "*extra quatuor maria*" for more than nine months preceding the birth (Blackstone). The present state of the English law on the subject appears to be that a child born during marriage is deemed illegitimate when, by good medical or other evidence, it is proved that it was *impossible* for the husband to be the father—whether from his being under the age of puberty, from his suffering from physical incapacity as a result of age or natural infirmity, or from the length of time which may have elapsed since he could have had intercourse, whether by reason of absence or death. With proof of non-access or immorality on the part of the mother, so important on these occasions, a medical witness is not in the least concerned. In a case of voluntary separation of husband and wife,[1] the children born are deemed to be the children of the husband, unless non-access is proved.

In *Atchley* v. *Sprigg*, [2] it was proved that husband and wife had lived together nine years without having had a child, and that they then separated, and did not live together again. Ten years after the separation, and while the wife was in the habit of committing adultery with another man, a child was born. This child was treated by the paramour as his own, was called by his surname, and was brought up by him. Its legitimacy was contested, and the Vice-Chancellor decided that notwithstanding the possibility of access on the part of the husband, the child was illegitimate. In this case there was conclusive evidence of illegitimacy, although access was possible.

[1] For decisions on the legal effect of separation deeds, see Rayden, *op. cit.* Ch. II

[2] 33 L. J. (Ch.), p. 345.

The presumption in favour of the legitimacy of a child born in wedlock, within the usual period of gestation, is a presumption which may be rebutted by admissible evidence that the husband could not possibly be the father[1]; but the rule of law that *neither the husband nor the wife* is permitted to give evidence of non-intercourse after marriage with the object of establishing that a child born during wedlock is not the child of the husband applies to divorce proceedings and to other proceedings instituted in consequence of adultery.[2] The fact of non-access, can, however, be proved by evidence *aliunde*.[3]

The adultery of the wife, although throwing the greatest doubt upon the paternity of the child, is not conclusive. It must be shown that the husband could not have had access which might result in paternity; otherwise the presumption of sexual intercourse between husband and wife, and consequently of legitimacy of the child, must prevail.[4]

Legitimation by subsequent Marriage. In England (by the Legitimacy Act, 1926) and in Scotland an illegitimate child is rendered legitimate by the subsequent marriage of the parents. If the father of the illegitimate person was at the time of the marriage domiciled abroad (including Scotland) by the law of which country the illegitimate person became legitimated by such marriage, the Legitimacy Act, 1926, enables such person to be recognised in England or Wales as having been legitimated from Janaury 1st, 1927, or from the date of the marriage, whichever happens last. The fact that the child was a bastard born in adultery is no obstacle, provided that subsequent marriage operates to legitimate such bastards by the law of the father's domicile at the time of the marriage.[5] Persons thus legitimated are entitled to succeed to property upon the intestacy of a parent as if such persons had been born legitimate.

By the Legitimacy Act, 1926, the legitimation dates from the commencement of the Act or from the date of the marriage, whichever happens last.

In determining legitimacy other than under the Legitimacy Act, 1926, the law of England has regard to the date of *birth*, which can be fixed, and not to the date of *conception*, which cannot be fixed; but a child born *after* the death of the wife, provided she had been lawfully married, is legitimate, although marriage is dissolved by death. Two cases are recorded in which living children were born after the death of the mother. The fact that the English law disregards the time, place, or date of conception may give rise to a singular question. A child may have been conceived before the marriage of the parents, and be brought into the world, by the Cæsarian operation, after the death of the mother: hence it would neither be *begotten* nor *born* in wedlock; and yet, according to the principles of the law of England, it would be the legitimate offspring of the marriage.

It happens sometimes that a child is born after the death of the husband. Conception is assumed to have taken place during wedlock; and, although the child is not born in wedlock, the presumption is in

[1] *Brierley* v. *Brierley and Williams*, [1918] P. 257; *Gaskill* v. *Gaskill*, [1921] P. 425.
[2] *Russell* v. *Russell*, [1924] A. C. 687.
[3] *Clarkson* v. *Clarkson* (1930), 46 T. L. R. 623 (naval officer's absence on service abroad).
[4] *Fosdike* v. *Fosdike and Hillier* (1925), T. L. R. 432; and *Warren* v. *Warren*, [1925] P. 107.
[5] *Collins* v. *A.-G.* (1931), 47 T. L. R. 484.

favour of legitimacy, unless non-access or physical incapacity be proved. Hence *conception* during wedlock and *birth* after the dissolution of the marriage by death—or conception before wedlock and birth during that state—or conception and birth during lawful wedlock, equally create a presumption in favour of the legitimacy of offspring.

Medical Evidence of Legitimacy

The medical evidence which may be required will refer to the following facts :—

Whether the woman has had a child
If so, whether the alleged child is of the age required } "Supposititious children."

If the woman has had a child, and no question is raised regarding substitution, its legitimacy must depend upon :—

On the part of the husband
1. Physical competency.
2. Access within reasonable time involving—
   (*a*) Natural duration of pregnancy.
   (*b*) Shortened ,, ,,
   (*c*) Prolonged ,, ,,

Whether the Woman has had a Child. This has been considered fully on pp. 45 *et seq.* The only matter which requires to be mentioned here for emphasis is that inasmuch as the child survived, it must have been of at least six months' intra-uterine development, and therefore the marks left by its birth have been and still may be manifest. On the other hand, it may be demonstrable that the woman is sterile (*vide* pp. 7 *et seq.*, also Vol. I, "Consent for Examination").

If so, whether the Child is of the Age Required. If a medical practitioner were always to be on his guard when called suddenly to a case of labour by persons who have not previously engaged him, there would rarely be any mistake in this connection.

A woman may substitute the living child of another woman for a dead child of which she herself has been delivered, or for a mole which may have passed from her. So, again, a male may be substituted for a female child, and *vice versâ*. When the question is simply whether delivery has or has not taken place, the calling in of a medical man would lead to discovery ; but if it is alleged that one *living* child has been substituted for another, the proof of this can depend on medical evidence only when the age of the supposititious child does not correspond to the date of the pretended delivery (*vide* "Age"). In a reported case it was proved that a woman had substituted a doll for the dead body of a child of which she pretended she had been delivered.

In *R. v. Mary Hall* the prisoner was accused of having conspired with a woman to make it appear that she had been delivered of a child. The nurse hired for the occasion was not allowed to be present during the alleged delivery, the prisoner acting as midwife. When the nurse was called into the room, she was shown by the prisoner the usual marks of a recent delivery, and on proceeding to wash the child, she found that it had already been washed, and was not a new-born unwashed child. It was then proved that the prisoner on the day of the pretended delivery had procured the child of another woman who had been attended by a medical man. A medical witness stated that he attended the woman after the alleged birth, and from what he saw he was sure that she had never given birth to a child.

A more daring attempt of this kind came before the House of Lords in the Wicklow Peerage case.[1]

The title and estates of the Earl of Wicklow passed at his death to his brother's issue. The first in succession was George Howard, who had died in October, 1864. He was married, in February 1863. In default of issue the estates devolved on his brother Charles, the second in succession. Mrs. Howard produced a male child, who, she alleged, was born on May 16th, 1864, and this child, if such were the case, would be the son of her husband George Howard, and consequently the rightful Earl. Mrs. Howard was at the time in lodgings, and the landlord and his wife and a sister of the latter, one, Rosa Day, were the principal witnesses for the claimant. Mrs. Howard was, or professed to be, taken suddenly ill at the date mentioned. The landlord went for a doctor, who was not at home, and on returning he was told that Mrs. Howard had been confined, and he saw an infant in Rosa Day's arms. This was the whole of the evidence of the child's parentage. Neither medical man nor nurse attended Mrs. Howard, although it was her first confinement, and the infant would have been a seven-months' child. It was never registered, and never baptized. There was evidence that Mrs. Howard had not borne a child, and that the child which she had produced as her own was obtained by her in August, 1864, from a girl who had been recently delivered in a workhouse. Mrs. Howard was clearly identified as the person who had taken away a child from the workhouse at this time. The House of Lords rejected the claim, and came to the conclusion that the witnesses had been guilty of perjury.

Suspicion arises in cases where a woman has not been attended by a medical man, and where there is an unexplained mystery as to the surrounding circumstances. A medical man may be called in to attend a woman some time after an alleged delivery, and unless he is careful, his name and reputation may be used as a shield to cover a gross imposture. In such a case he should take nothing for granted ; but should insist upon having a knowledge of all the facts, and should see all the parties alleged to have been present at the delivery. He must not trust to the appearances of blood in the room or the appearances of a burnt placenta, for these conditions may be easily imitated by an artful midwife.

Physical Competency of the Husband. This has already been fully considered (*vide* "Impotency," p. 2). It calls for no further observation beyond a caution as to cryptorchism, *q.v.*

Duration of Pregnancy.[2] For medical purposes we have only to consider the duration of natural pregnancy. (For a full discussion of the average and its limits either way *vide supra*.) All that need be stated here is that the **period of gestation is not fixed by law.** In all cases of contested legitimacy, the question respecting the duration of gestation, when it arises, is left entirely open by the law of England. The decision of a court of law would be founded *quoad* the duration of pregnancy on the opinions of experts selected for the occasion, and each case would be decided on its own merits. Precedents do not assist on these occasions, because a court may, on non-medical grounds, declare legitimate a child born in the thirty-eighth week of gestation, whereas in another case a child may be declared legitimate where birth had taken place in the forty-third week. In some cases *forty* weeks (or 280 days), and in others forty-three weeks (or 301 days), have been taken as the *ultimum tempus pariendi*.

[1] *The Wicklow Peerage Case*, Com. for Privileges, April 1st, 1870.

[2] For an excellent paper on this subject by T. W. Eden, M.D., see *Trans. Med.-Leg. Soc.* for 1922–23.

The question of the duration of pregnancy has been raised in many cases of which the following are typical :—

In *Cotterall* v. *Cotterall*, a child was born during the marriage, and the husband petitioned for a divorce on the ground of adultery. In order to have been the child of the husband, the birth must have taken place after *twelve months'* gestation. The husband left his wife in New South Wales, and was absent for that period of time without possibility of access. The judge (Lushington), without entering into the question of protracted gestation, upon proof of this allegation, at once pronounced for the divorce.

Such a duration of pregnancy is not supported by any known facts, and is altogether opposed to medical probability.

In *Bowden* v. *Bowden*,[1] the period of gestation was 307 days.

One of the most interesting cases on legitimacy is the Gardner Peerage case which came before the House of Lords early in the nineteenth century. A full account of the medical evidence was published by Lyall.[2]

Alan Legge Gardner, the son of Lord Gardner by his second wife, petitioned to have his name inscribed as a peer on the Parliament Roll. The peerage was, however, claimed by another person, Henry Fenton Jadis, *alias* Gardner, who alleged that he was the son of Lord Gardner by his first and subsequently divorced wife. It was contended that the latter son was illegitimate ; and in order to establish this point, the evidence adduced was partly medical and partly moral. Lady Gardner, the mother of the alleged illegitimate child, parted from her husband, on board of his ship, on January 30th, 1802. Lord Gardner went to the West Indies, and did not again see his wife until July 11th following. The child whose legitimacy was disputed was born December 8th of that year. Therefore the plain medical question, taking the extreme view, was whether a child born 311 days (*forty-four weeks and three days*, from January to December) or 150 days (*twenty-one weeks and three days* from July to December) after possible intercourse could be the child of the husband, Lord Gardner. If these questions were answered in the affirmative, then it followed that this must have been either a premature or a protracted birth. There was no pretence that it was a premature case, the child having been *mature* when born. The question then was reduced to this—Was this alleged protracted gestation of 311 days consistent with medical experience ? Seventeen leading obstetric practitioners in the kingdom gave evidence which was conflicting, but a majority agreed that natural gestation might be protracted to a period which would cover the birth of the alleged illegitimate child. It was proved that Lady Gardner, after the departure of her husband, was living in open adulterous intercourse with one, Jadis ; and on this ground Lord Gardner obtained a divorce. He subsequently married a second wife, by whom he had the claimant, Alan Legge Gardner. It was contended that the other claimant was really the son of Lady Gardner by Jadis. The decision of the House was that this claimant was illegitimate, and that the title should descend to the son of the second Lady Gardner.

On moral grounds the decision could not be impugned ; but, medically, it assumed that gestation could never be protracted to the 311th day after probable intercourse. Inasmuch as conception is not necessarily the immediate result of intercourse, and as we have no data for fixing the precise time of its occurrence, this decision could hardly be supported on medical grounds. We should not be justified in affirming that every child born forty-four weeks and three days after the opportunity of intercourse with the husband was *ex necessitate rei* an illegitimate child. Of the seventeen medical experts who gave evidence, five supported the opinion that the duration of human pregnancy was limited to

[1] (1917), 62 Sol. J. 105.

[2] "Med. Evid. in Gardner Peerage Case," 1827.

about nine calendar months, *i.e.*, from thirty-nine to forty weeks, or from 273 to 280 days—or, strictly speaking, from 270 to 280 days; one of the witnesses, indeed, said from 265 to 280 days. These witnesses, of course, gave a negative to the possibility that Henry Fenton Jadis, *alias* Gardner, could have been the product of 311 days' gestation. On the other side, of twelve medical men who seemed to agree respecting the above-mentioned period as the natural term of gestation, the greater number maintained the *possibility* of pregnancy being protracted to nine and a half, ten, or even eleven calendar months, and of course to 311 days —the alleged term of gestation at which the counter-claimant was born — and they thus admitted the possibility that H. F. Jadis, *alias* Gardner, might be a ten-and-a-half-months child.[1]

In a well-marked instance of gestation from a single intercourse, noticed by Reid, the interval was 293 days; and by referring to the case of *Rigby* and *Merriman*, it will be seen that the periods of gestation from a single intercourse have varied to a much greater degree than the two here placed in comparison. The following case was tried in the United States:—

The defendant was indicted for fornication and bastardy. The prosecutrix, aged twenty-three, stated that she had had intercourse with the defendant on September 24th, 1842, and with no other person before or subsequently. She was delivered of a child on August 7th, 1843, *i.e.*, after 317 days, or *forty-five weeks and two days' gestation*; and she swore that the defendant was the father of the child. The menses ceased about three weeks after intercourse, and they only appeared again slightly about five weeks before the child was born. At this time she had pains which continued more or less until her delivery. She first knew that she was pregnant three or four weeks after intercourse.

The defence was that, from the period of time which had elapsed, the defendant could not have been the father of the child. He therefore merely proved his absence and did not return until after the birth of the child. No evidence was adduced to impeach the character or conduct of the woman. It was proved that she had always borne a good reputation, and that she had been seduced by the defendant under a promise of marriage. Rodrigue deposed that in a practice of nineteen years, he had attended some hundreds of cases of midwifery, and the longest period of gestation which he had known was *ten months*. He considered the pains described by the prosecutrix to have been the commencing pains of labour. The court charged the jury strongly in favour of the medical testimony on protracted gestation, and they returned a verdict of guilty, thereby finding that the defendant was the father of the child. It transpired that a wife of one of the jurymen had during one pregnancy gone ten months.

In another case in the United States there was reason to believe that gestation had continued for a period of 320 days.

In a seduction case the parties met in Guernsey, and it was admitted that an intimacy had existed between them. The defendant left the island on April 15th, and did not return to it. The plaintiff was delivered of a child on February 15th following, *i.e.*, 307 days or forty-four weeks less one day, after the departure of the defendant. There was no evidence that the plaintiff, either before or subsequently, had had connection with any other man.

For the defendant, it was contended that he could not possibly have been the father of the child, inasmuch as, in that event, there must have been a period of gestation of over 300 days, which, it was contended, was physically impossible. Two medical witnesses for the plaintiff said

[1] Lyall's "Med. Evid. on the Duration of Pregnancy," p. 8.

that though 275 days was the usual period of gestation, they had known cases of 297 or 300 days. Two medical witnesses for the defence stated that in their belief the current of medical opinion ran now strongly in an opposite direction, and tended to limit rather than to extend the limits of possible gestation ; and though they would not say that it was absolutely impossible that the period should extend to 300 days, they believed it to be so improbable as to be practically incredible. The plaintiff was awarded damages. The medical witnesses on both sides agreed that the gestation might have been protracted to the extent which would have made the defendant the father of this child.

In the case of *Gaskill* v. *Gaskill*,[1] in which adultery by the wife was alleged, the period between the last date of possible access by the husband and the date of birth was 331 days. There was no evidence of misconduct by the wife, and the husband's petition was dismissed, with the necessary inference that the child born was legitimate.

The practice in contested cases of legitimacy consists in establishing the possibility of access on the part of the husband. In order to rebut the legal presumption of access, where husband and wife are living near each other, evidence of something more than mere probability of non-intercourse must be adduced. When possibility of access is proved, the medical question arises, whether the term of gestation falls within the limits assigned by the best medical experience. In *Gaskill* v. *Gaskill*,[1] three eminent specialists in obstetrics and gynæcology gave evidence as to the length of gestation.

PATERNITY

Disputed Paternity. Parental Likeness.[2] It has been stated that the law does not pretend to determine who begat a child when it has been born during wedlock. But medical witnesses have recommended that family likeness should be considered from several aspects—not merely a likeness in *feature* and figure, but in gesture and other personal peculiarities which may have characterised the alleged parent. These are called questions of *paternity* : they seldom occur except in reference to cases of bastardy, and when they do present themselves, the evidence thus procured, even if affirmative, is properly regarded as only corroborative. In the Townshend Peerage case,[3] a presumption based on family likeness was admitted. The person whose legitimacy was in question was sworn by one of the witnesses to have shown such a likeness as a child to the alleged adulterer that he would have known him among five hundred children.

The proceedings in the Douglas Peerage case (1767 9) show that evidence of this kind is occasionally of some importance.

The peerage was claimed by Archibald Douglas—the survivor of two brothers after the death of the alleged parents, Sir John and Lady Douglas. The claim was disputed, on the ground that the claimant and his deceased brother were supposititious children. Evidence for and against the legitimacy of the claimant had been collected from every quarter, and after it had been most minutely sifted

[1] [1921] P. 425.

[2] For a paper on the "Evidence of Resemblance in Paternity Cases," by Sir Francis Newbolt, K.C., see *Trans. Med.-Leg. Soc.* for 1923–24.

[3] House of Lords, May, 1843.

and criticised, the case came on for judgment, in the Court of Session in Scotland, on July 7th, 1767. So important was the cause deemed, that the fifteen judges took eight days to deliver their opinions.

The result was that seven of the judges voted in favour of the identity or legitimacy of Archibald Douglas, *alias* Stewart, and seven against it; the Lord President, who had the casting vote, agreed with the latter, thus furnishing one among numerous instances that judges as well as physicians can differ with precisely the same facts before them. An appeal from this decision was taken to the House of Lords by which the judgment of the Court of Session was reversed in 1769, and Archibald Stewart (or Douglas) declared to be the son of Lady Jane, the sister of the previous holder of the title. Much stress was laid, in favour of the legitimacy of these children, on the fact that they closely resembled—the one Sir John and the other Lady Douglas. The resemblance was said to be general: it was evident in their features, gestures, and habits. Lord Mansfield, in delivering judgment, said:—

"I have always considered likeness as an argument of a child being the son of a parent, as the distinction between individuals in the human species is more discernible than between other animals. A man may survey ten thousand people before he sees two faces exactly alike; and in an army of a hundred thousand men every man may be known from another. If there should be a likeness of features, there may be a difference in the voice, gesture, or other characters, whereas a family likeness runs generally through all of these; for in everything there is a resemblance, as of feature, voice, attitude, and action."

This kind of evidence has been strongly objected to from its uncertainty; and it was in this instance much disputed whether one of the children resembled Lady Douglas, but it seems to have been generally admitted that the other child resembled the husband, Sir John. Evidence from family likeness is not strictly medico-legal; it can be furnished only by friends and relatives who have known the parties well, and are competent to speak from personal knowledge. The affirmative evidence in such cases will be stronger than that which is negative, for it could hardly be inferred that a person was illegitimate because he did not resemble his parent.

The following extract[1] is of interest with reference to the modern legal view of the value of physical resemblance as evidence in cases of disputed paternity:—

"The Scottish law courts have once more rejected evidence of physical resemblance between child and alleged parent in a case where paternity is in dispute. In preliminary proceedings in his claim to the Seafield peerage Mr. Alexander Grant, who seeks a judicial declaration that he is the eldest lawful and legitimate son of the seventh Earl of Seafield, proposed to offer evidence that not only he himself but also his daughter and other members of his family resembled his alleged parents in feature, gait, and personal mannerisms. Lord Ashmore made an interlocutory order excluding these averments; from that order Mr. Grant appealed, but the Second Division of the Court of Session has (according to a report in *The Times* of December 19th) dismissed his appeal with costs. It will be recalled that ten years ago in the Slingsby case an attempt was made to establish in the English High Court the legitimacy of a boy of

[1] *Lancet*, December 26th, 1925.

four years of age by offering evidence *inter alia* of physical similarities between the child and his reputed parents. On appeal the House of Lords did not look with much enthusiasm upon evidence of this kind ; Lord Shaw in particular, speaking with the authority of a Scottish lawyer, pointed out that in Scotland such evidence had been rejected for more than a century on the ground that it was ' loose and fanciful,' and added that in England it was open to the same objection. In the Seafield peerage claim Lord Anderson repeated Lord Shaw's two epithets, while the Lord Justice Clerk commented on the remoteness of investigating alleged resemblances unto the third and fourth generations. It was proposed, he said, to find resemblances to her grandfather in a granddaughter. When he surveyed the vista opened up by these averments, he thought, in Professor Bell's phrase, that the situation would make wild work of the law of legitimacy. English law is not quite so rigid as Scots law in excluding evidence of this kind. It will be remembered that photographs were produced in the Russell divorce case to suggest the resemblance of baby to father, and that in the Slingsby case Sir George Frampton, the sculptor, acted as a kind of informal assessor in assisting the judge to detect details of physical similarity. English law seems to allow that, in cases of disputed paternity, the phenomenon of facial resemblance between parent and child is one which a jury may take into account and may weigh along with other evidence. But even English law must draw the line somewhere, and if in an English court it was sought to trace this resemblance through three or four generations, the judge, if he did not exclude the evidence altogether, would doubtless warn the jury not to pay much attention to it."

Parental likeness may be occasionally indicated by colour or peculiarities belonging to the varieties of mankind, as in intermarriages with Negro or Mongolian. In such a case the evidence afforded becomes much stronger ; and supposing that two men of different nationalities have intercourse about the same time with the same woman, the colour of the skin of the offspring might possibly enable a court to determine the question of paternity. On more than one occasion a black woman has given birth at the same time to a black child and a mulatto. A case is recorded in which a negress gave birth to twins, one of whom was black and the other white. This was probably a case of superconception. In a seduction case the defendant was a man of colour, and the child born of the alleged adulterous intercourse was born coloured and with woolly hair. The husband and wife were both light. This peculiarity fixed the paternity of the child on the defendant.

Personal Deformities are not necessarily transmitted from parent to child ; yet it would appear that a disputed question of affiliation has been settled on this principle. The claim in an affiliation case rested chiefly on the fact that the child had been born with five fingers and a thumb on the right hand, the putative father having been born with a similar malformation on both hands. It was argued, on the other side, that the deformity might have arisen from the mother's imagination, as, while pregnant, she was constantly in the habit of seeing the defendant. The magistrates decided that he was the father of the child. This man might have been the victim of a coincidence. Six-fingered children are, it is well known, born occasionally of five-fingered parents : and as the

deformity existed only on one hand in the child, while it was on both hands in the parent, the medical proof that it was actually transmitted by generation was not clearly made out. In some instances attempts have been made to fix the paternity of the child by the *colour of the hair*, but this evidence is far less conclusive than that afforded by the colour of the skin.

In another case it was alleged that the wife of the plaintiff had had intercourse with the defendant, and that the last two children were the offspring of the latter. The plaintiff and his wife had dark hair, as well as all the children with the exception of the last two, both of whom had red hair; the defendant had red whiskers and sandy hair.

No particular stress was laid upon this evidence, but it was received as a kind of indirect proof. Not much confidence can be placed in facts of this description, since red-haired children are often born to parents who have dark hair; and in one case the children born in wedlock were observed to have dark and red hair alternately.

Eye colour is another factor which may be used in determining parentage. It is stated[1] that blue eyes are recessive to dark eyes, and that blue-eyed parents always have blue-eyed children.

The Blood Group Test in Disputed Paternity. The determination of the blood group of the child and the alleged parents may enable the observer to give definite evidence about the possibility of a certain person being the father of a child or of deciding, when two persons are involved, which of the two if any could be the father. In modern times evidence of non-access (which cannot be given by either of the spouses) can be given by a scientist who carried out a blood test on the spouses and the child. In *Wilson* v. *Wilson*[2] evidence was given that the husband's group was OM, that the wife's was BM, and that the child's was ABN. The Court held that the husband was not the father of the child, and granted a decree of nullity. For details of this test the reader is referred to p. 417, Vol. I. For references to the Rhesus factor, see pp. 419 and 423 of Vol. I, and the Addendum to this Volume.

Affiliation. Questions of paternity are involved in affiliation cases. A man may allege that he is not the father of a particular child, by reason of certain circumstances upon which a medical opinion may be required.

Two men, A. and B., had intercourse, unknown to each other, with a young woman of delicate health, and after this had continued for some years she was delivered of a female child—nine calendar months and three days after sexual intercourse with A., and nine calendar months, less five days, after similar intercourse with B.; or at the end of 279 days after intercourse with A., and at the end of 271 days after intercourse with B.—that is, a period of *eight days* elapsed between the periods of intercourse of the two men. The woman had no menstrual discharge in the meantime, and it is not believed that she knew any other man; she went her full time, had a good labour, and produced a fine healthy girl; she had a plentiful supply of milk, and enjoyed better health during and after her pregnancy than at any other time. The woman died, and the circumstances of the mixed intercourse having become known to A. and B., they both refused to maintain the child. A. contended that, as the woman was not delivered until nine months and three days after the connection with him, it was physically

[1] "Studies in Human Inheritance," Snyder, *Jour. Amer. Med. Ass.*, February 19th, 1927.
[2] *Lancet*, 1942, I. 570.

impossible that the child could be his. B. contended, on the other hand, that 280 days, and not nine months, is the period of gestation ; and that the child having been born 279 days after connection with A., and only 271 days after connection with B., it was therefore probable that the child was begotten by A. There was no perceptible likeness to either of the men in the child, but a marked likeness to the mother.

The periods of 271 and 279 days are comprised within the ordinary range of gestation : hence there would be no *medical* ground for affiliating the child to one man more than the other. The length and weight and other features relating to the child showed that it was mature, and had reached its full development, but this would not suffice to justify a medical opinion on the precise date of conception. Such a question does not admit of any definite answer in reference to paternity, the dates of intercourse being too close together.

When two men have intercourse with the same woman on the same day, it is impossible to settle the paternity except by the accident of likeness or by the blood group test. In cases of affiliation under the law of bastardy, the evidence of the mother, if corroborated, is received in support of a question of disputed paternity ; sometimes these cases are decided by the length of the period of gestation. A man may prove, or a woman may state, that the intercourse took place at such a remote period as to be inconsistent with the ordinary duration of pregnancy.

Medical questions may also arise whether gonorrhœa or syphilis are invariably transmitted by intercourse or whether they interfere with the act of procreation. In ordinary circumstances both questions must be answered in the negative.

Posthumous Children. A case involving a question of paternity may present itself on the marriage of a widow soon after the death of her first husband. If a child were born before the expiration of ten months, it might be a question whether it was a child of the first or second marriage —of the dead or the living husband ; and although there might be no dispute concerning its legitimacy, yet it would be difficult to determine its *paternity*. Such a case appears hypothetical. In order that any doubt should exist, a woman must marry within, at the furthest, *six weeks* after the death of her first husband, or the birth of the child would fall beyond the furthest limit of gestation, so far as he was concerned. If there were a greater likeness to the first than to the second husband, this fact alone would not be sufficient to rebut the legal presumption of the real parentage of the child. Evidence much stronger than this would be required for such a purpose.

CHAPTER VII

RAPE

This subject will be considered under the following heads : –

Definition of the crime.
Age relations of rape.
Mental condition of victim.
Time for bringing an action.
Special circumstances in which rape may take place.
Medical aspects of rape.
Rape on infants and children up to the age of sixteen.
Rape on women and girls over sixteen.
Disappearance of evidence.
False charges of rape.
Pregnancy following rape.
Rape on the dead.
Rape by females on males.

Definition of the Crime

Rape consists in having unlawful carnal knowledge of a woman by force and against her will and, in Scotland, of a girl below twelve, whether by force or not. In England for many years the crime was punished as a capital crime, but penal servitude was substituted by the Offences Against the Person Act, 1861. By this Act (s. 48) it is enacted that—

"*Whosoever shall be convicted of the crime of rape shall be guilty of felony, and being convicted thereof, shall be liable, at the discretion of the court, to be kept in penal servitude for life.*"

A charge of rape may be prosecuted at any time, and notwithstanding any subsequent consent of the woman alleged to have been ravished.[1]

By the Criminal Law Amendment Acts 1885 to 1928, certain conditions are laid down as regards age, mental conditions, etc.

Degree of Penetration Necessary to Constitute Rape. The crime is committed if there is proof of penetration : it is not necessary to prove actual emission.[2] In *R.* v. *Russen*,[3] it was held that a degree of penetration so slight as not to injure the hymen would be sufficient in law for the completion of the crime. In the case referred to, the hymen of the young girl was proved to be entire. Under the direction of the judge,

[1] *R.* v. *Osborne*, [1905] 1 K. B. 551.
[2] Offences against the Person Act, 1861, s. 63; *R.* v. *Marsden*, [1891] 2 Q. B. 149.
[3] 1 East, P. C. 439.

the prisoner was convicted. A sufficient degree of penetration to constitute rape may take place without necessarily rupturing the hymen[1]; but in a special case there must be medical evidence to show that there was actual penetration—the degree of penetration being quite immaterial. It is true that there could not be a complete introduction of the adult male organ into the vagina of a child without rupture or laceration of the soft parts; but the absence of such marks of violence would not justify a medical witness in denying the perpetration of the crime, since the law does not require proof either of a complete or of a violent introduction. Penetration to the vulva is sufficient to constitute the crime.

In one case the evidence left no doubt that the accused had had carnal knowledge of a girl about ten years old. The surgeon stated that there were considerable marks of violence about the pudendum, but completion (*i.e.*, complete penetration) was, in his opinion, physically impossible on a child under ten years of age. Upon this evidence, the charge of felony was abandoned.

In another case the child was older, but the facts are relevant.

A man was charged with rape upon his own child, a girl fourteen years of age. The child was examined about two days after the alleged rape, but no injury about the vulva or adjacent parts was found, and the hymen was unruptured. One medical witness was of opinion that no rape had been committed; but two other medical witnesses, men of experience and integrity, stated their belief that the crime had been perpetrated. The latter had examined the child soon after the alleged offence, and a day or two before the first witness examined her. The accused was acquitted of rape, but found guilty of assault.

The absence of any marks of injury about the vulva so short a time after the alleged criminal act, and the fact that the hymen was unruptured, justified in some measure the opinion of the first medical witness that there was no medical proof of rape. At the same time he candidly restricted his opinion by saying, that if by rape is meant penetration to the vulva, then it was effected; but there was no evidence to show vaginal penetration—on the contrary, the unruptured state of the hymen in an alleged forcible intercourse was against this view. The statute says nothing about the rupture of the hymen as a necessary part of the evidence; the law merely requires penetration; this may occur, and the hymen remain intact. Vulval penetration, whether with or without violence, is as much rape as vaginal penetration.

In Scotland this question came before the courts in the case of Macrae where the defence was that there must be proof of full and complete penetration, and that there was not sufficient evidence to show that penetration had taken place into the canal of the vagina beyond the vulva. The judge directed the jury that the evidence of the accused's guilt was complete; that in a charge of rape scientific and anatomical distinctions as to where the vagina commenced were worthless; and that by the law of Scotland it was enough if the woman's body had been entered. In a case where there was no evidence of emission, and the girl was young, it was not necessary to show to what extent penetration had taken place, or to prove that it had gone either past the hymen, or even so far only as to touch the hymen. The accused was convicted.

[1] *R.* v. *Hughes*, 2 Mood. C. C. 190.

Age Relations of Rape

In the Male. A boy under the age of *fourteen years* is presumed in law to be incapable of committing a rape[1]; but he can be convicted as a principal in the second degree if it be proved that he was present aiding and abetting the principal offender. In a case in which a boy aged 14 was charged with rape, the judge directed an acquittal. Although in other felonies sometimes *malitia supplet ætatem*, yet as to this particular act, the law presumes him to be impotent. Recorded cases, however, show that boys of this age are not always impotent. Instances of precocious puberty are, it is well known, very frequent. As proof of emission is not necessary in order to complete the crime, it may become a technical question whether, admitting the existence of guilty knowledge, the crime might not be completed in law long before the signs of puberty were fully developed. This question is very likely to arise where boys are charged with the crime of sexual assault upon female infants. The proof must depend upon the medical evidence.

In the Female. "*Any person who unlawfully and carnally knows any girl under the age of thirteen years shall be guilty of felony.*

"*Any person who attempts to have unlawful carnal knowledge of a girl under the age of thirteen years shall be guilty of a misdemeanour.*

"*Any person who unlawfully and carnally knows or attempts to know or to have unlawful carnal knowledge of a girl being of or above thirteen but under sixteen years of age shall be guilty of a misdemeanour.*"[2]

For determination of these ages and the medical evidence thereon, *vide* Vol. I., "Age".

It will thus be seen that a girl under thirteen is presumed not to be capable of either giving or withholding her consent.

By the Criminal Law Amendment Acts, 1885 to 1928, it is no defence to a charge or indictment for an indecent assault on a child or young person under the age of sixteen to prove that he or she consented to the act of indecency; and by sect. 2 (as amended by the Act of 1928) it is enacted that reasonable cause to believe that a girl was of or above the age of sixteen years shall not be a defence under sects. 5 or 6 of the Criminal Law Amendment Act, 1885, provided that no prosecution shall be commenced more than twelve months after the commission of the offence, and provided that in the case of a man of twenty-three years of age or under the presence of reasonable cause to believe that the girl was over the age of sixteen years shall be a valid defence on the first occasion on which he is charged with an offence under this section. The exception applies to a man until he reaches his twenty-fourth birthday.[3] As to proof of the accused's belief, see *R.* v. *Harrison and others*.[4]

Mental Condition of Victim in Rape

"*Any person who unlawfully and carnally knows or attempts to have unlawful carnal knowledge of any female idiot or imbecile woman or girl, under circumstances which do not amount to rape, but which prove that the*

[1] 1 Hale, P. C. 631; *R.* v. *Waite*, [1892] 2 Q. B. 600.
[2] Criminal Law Amendment Act, 1885, s. 4.
[3] *R.* v. *Chapman*, [1931] 2 K. B. 606.
[4] [1938] 3 All E. R. 134.

offender knew at the time of the commission of the offence that the woman or girl was an idiot or imbecile, shall be guilty of a misdemeanour."[1]

There is no specific mention of age here, but the insertion of the words "girl" and "woman" suggests that the mental condition is meant to be an additional preventive against the crime, age being already sufficiently safeguarded.

Special Circumstances in which Rape may take place or be alleged to have taken place

Under the Influence of Narcotics or Alcohol. If a woman be drunk or asleep from drink or other narcotic, it is possible for sexual intercourse to take place without her consent.

If a woman alleges that prior to the rape she was rendered unconscious by drink, or that a soporific was administered in such drink, an early examination should be made to ascertain whether there are any signs indicating the ingestion of a specific narcotic such as morphine or of alcohol. Careful enquiry should be made into the circumstances in which the drink was taken, whether it was taken freely or forced upon the woman, the quantity taken, the manner and rate of onset of symptoms and how much of the assault is remembered. All these questions and others that would be sure to arise from her explanations, require to be carefully considered before an opinion on the case can be arrived at. If it were proved that a known soporific, such as opium, had been used, there would be little doubt about a conviction; but, if no foul means had been used, there is a definite probability that a constructive consent had been given, however much it may have been regretted afterwards. The question (apart from the nature of the drug) is particularly one for a jury rather than for scientific evidence.

In the case of *H.M. Advocate* v. *Grainger and another*[2] a ruling was given that by the law of Scotland sexual connection with a woman found in a state of insensibility from intoxication did not constitute the crime of rape. In that case it was argued that since rape was defined as the carnal knowledge of a woman by force and against her will, the crime could not be committed unless the woman was in a condition to exercise her will-power and offer resistance. What was said to have been done by the accused was not rape but was a criminal offence, viz., the crime of inflicting clandestine injury on a woman, and must be indicted as such and not as rape.

In the case of *H.M. Advocate* v. *Logan*[3] a ruling was given that if drink were administered by the accused with the object of overcoming the resistance of the woman the crime might be rape, but if the drink had been taken by the woman of her own free will the crime so far as it was possible to define it came within the category of indecent assault.

Under the Influence of Anæsthetics. The vapours of ether and chloroform have been criminally used in attempts at rape, although by the Criminal Law Amendment Act, 1885, the administration of any drug, matter, or thing to a girl or woman with intent to stupefy or to overpower, so as thereby to enable any person to have unlawful carnal connection with such woman or girl, constitutes a misdemeanour.

[1] Criminal Law Amendment Act, 1885, s. 5 (2).
[2] *Scots Law Times*, (1932) 28.
[3] *Scots Law Times*, (1937) 104.

In a case which occurred in France, a dentist was convicted of a rape upon a patient to whom he had administered the vapour of ether. The patient was not completely unconscious, but she was rendered wholly unable to offer any resistance. A dentist was convicted of rape in somewhat similar circumstances in the United States; but it was thought that the woman had made the charge under some delusion. In a Liverpool case it was alleged that a dentist rendered a woman patient suddenly insensible by chloroform, and then had intercourse with her. In cross-examination, however, it transpired that the woman was not rendered insensible at all, but was conscious of all that was going on, and that she might have given an alarm, but did not.

Many stories of this kind, when carefully investigated, will be found inconsistent and untrue. It is not the property of chloroform or of any narcotic substance, in a non-fatal dose, to render a person instantaneously insensible and powerless. If given in a non-fatal dose the effects are slowly and gradually produced; if they are produced in a few minutes the dose must have been large, in which event death would probably take place. Many of the charges made against medical men and dentists by women who allege that they have been violated whilst under the influence of anæsthetics are false. Anæsthetics stimulate the sexual functions and the ano-genital region is the last to give up its sensitiveness. Such charges are sometimes made in all good faith by modest women. A woman under the partial influence of an anæsthetic may mistake the forcible attempts to restrain her movements, while she is passing through the preliminary stage of excitement induced by the anæsthetic, for an attempt upon her chastity. A woman engaged to be married visited her dentist accompanied by her fiancé: chloroform was given, and a tooth was extracted in his presence; but she could hardly be convinced that the dentist had not made an attempt upon her chastity.

These anæsthetic cases inculcate the golden rule from which no exception should be made in any circumstance whatever, *viz.*, **that a medical man should never administer an anæsthetic without there being present at least one other disinterested person in addition to himself and the patient.** It is much easier for a charge of this sort to be brought forward than to be disproved, and especially so if there be a conspiracy to blackmail.

On the other hand, the woman may not take the anæsthetic voluntarily, but may allege that she was anæsthetised by violence or while asleep. That a person can be anæsthetised without being awakened from a natural sleep is certainly possible, though in the many cases in which the experiment has been tried it has failed more frequently than it has succeeded; a statement, therefore, to this effect when made by the victim of an alleged rape, should require a good deal of corroboration. That a woman may become paralysed, or may even faint, from fear, and subsequently be anæsthetised against her will, is also certainly possible; but the allegation that a handkerchief saturated with chloroform produced instant unconsciousness when thrown over the face requires very careful consideration and close cross-examination as to fear, and what followed immediately upon the covering of the face; because chloroform anæsthesia, or anæsthesia by any other drug, cannot be thus instantly produced. In cases such as these, signs of a struggle and consideration of the physical and moral and emotional character of the woman and man respectively become the principal factors.

Under the Influence of Fear or other Moral Restraint or Deception. Some medical witnesses maintain that rape cannot be perpetrated by one man alone on an adult woman of good health and vigour; and therefore they regard all accusations made in these circumstances as false. Whether a rape has been committed or not, is a question of fact for a jury and not for a medical witness. The fact that the crime has been perpetrated can be determined only from the evidence of the prosecutrix and of other witnesses; nevertheless a medical practitioner may be able to point out to the court circumstances which might otherwise escape notice. Apart from the cases of infants, idiots, persons of unsound mind, and weak and delicate or aged women, it does not appear probable that intercourse could be accomplished against the consent of a healthy adult, under conditions of fear alone, unless she fell into a state of syncope, from terror and exhaustion. Inability to resist from terror, or from an overpowering feeling of helplessness, as well as horror at her situation, may lead a woman to succumb to the force of a ravisher, without offering that degree of resistance which is generally expected from a woman so situated.

When several act in combination against a woman the case is rendered much more simple, and resistance may be impossible; but in this case we may expect to find some marks of violence on her person, if not on the genital organs.

A woman may yield to a ravisher, under threats of death or duress; in this case her consent does not excuse the crime; but this is a legal rather than a medical question. An aged woman can scarcely be expected to resist a strong man. Chevers mentions a case in which a man was convicted of rape and an aggravated assault on a woman of *seventy* years of age.

Two youths, each *æt.* 16, were tried for the rape of a girl *æt.* 14, but who appeared somewhat older. It was alleged that the girl was seized by the arms by N. and held against some palings, whilst G. had connection with her, she being in the standing posture. She then ran away; but was pursued and was seized by the arms by G. whilst N. now had connection, standing. The girl went home agitated, but made no complaint to her mother, who next day washed the girl's under-linen, but observed nothing unusual. When medically examined six days after the occurrence, the vagina was dilated and inflamed, and the hymen ruptured and healed. The connection was not denied, the defence being that the girl, who had been sliding on the ice with the boys, was an inviting party. There was an acquittal on the charge of rape and a conviction for intercourse with a girl under sixteen years of age.

In *R.* v. *Beard*,[1] the House of Lords decided that, except in cases where drunkenness amounts to insanity, where a specific intent is an essential element in an offence, evidence of a state of drunkenness rendering the accused incapable of forming such an intent should be taken into consideration with other facts proved with a view to determining whether the accused had formed the intent necessary to constitute the particular crime; but evidence of drunkenness falling short of a proved incapacity to form such intent did not rebut the presumption that a man intends the natural consequences of his acts. In cases of rape, therefore, drunkenness is no defence to murder following rape, unless it be proved that at the time of committing the rape the accused

[1] [1920] A. C. 479.

was so drunk as to be incapable of forming the intent to commit it, inasmuch as the death resulted from a series of acts, the rape and the act of violence, causing suffocation, which could not be regarded independently of each other. The rule in *R.* v. *Meade*[1] appears to be confined to cases where it is necessary to prove a specific intent.

In the *Lancet*, 1902, vol. 1, p. 175, will be found an account of a case in which religious fervour was the means used by a man to effect his purpose; the case known as the Horos case aroused much indignation at the time; the man pleaded impotence, which was disproved.

In another case the prisoner advertised for girls for the stage, and under the pretence of teaching them to "do the splits" he had connection with at least two girls, *æt.* 19 and 23.

In all such cases the age and mental condition (innocence) of the victim are the principal factors in determining the guilt of the accused.

A consent or submission obtained by fraud is not a defence to a charge of rape. In *R.* v. *Williams*[2] the accused ravished a girl of sixteen years on a pretence that she was being treated medically.

4. **Can a Woman be Violated during Natural Sleep?** It may be a question whether a man can have intercourse with a woman without her knowledge while in a state of *unconsciousness from natural sleep.*

Casper met with a solitary case in which a girl, *aet.* 16, accused a man of having had intercourse with her while she was sleeping in her bed. She asserted that she was not conscious of his action until he was in the act of withdrawing from her. On her own statement she was *virgo intacta* up to the date of the occurrence. Upon the facts of the case, Casper came to the conclusion that, if her statement was true, the man could not have had intercourse with her without causing pain and rousing her to a consciousness of her position. The hymen was not destroyed, but presented laceration in two places. This and other facts showed that there had been intercourse, but did not prove that this had taken place without the consciousness of the woman.

The following case goes to show that it is possible, in certain circumstances, for this crime to be perpetrated during sleep:—

A respectable married woman, who had had children, threw herself on her bed with her clothes on, late one evening, and fell fast asleep. She was first awakened by finding a man upon her body, in the act of withdrawing from her. This man, a servant in the house, was given into custody on a charge of rape. In the first instance he did not deny the act, and there was no reason to believe that the prosecutrix was aware of the accused's conduct until the crime was completed, and she was awakened in the manner described—apparently by the weight of the accused's body. The accused was convicted.[3]

A case which may serve to throw a little light upon this question is reported by Casper. A married woman alleged that a man had had intercourse with her while in bed, and when she was asleep. She admitted, however, that she was conscious that someone was lying upon her, and that she asked who it was, showing, as Casper remarks, that she had a knowledge of what was going on, and that she had a *doubt* whether the person was her husband.

[1] [1909] 1 K. B. 895.
[2] [1923] 1 K. B. 340.
[3] *Edin. Month. Jour.*, December, 1862, p. 570.

In regard to the question whether it is possible to commit rape while the victim is asleep, a majority of the Scottish judges decided, in the case of Sweenie that feloniously having connection with a sleeping woman was not indictable under the name of rape, inasmuch as, apart from the force implied in the act of connection, there was no force used to overcome the will of the woman. But they held, however improbable it might be, it was quite possible that a man might have connection with a sleeping woman, forgetting, possibly, "*non omnes dormiunt qui clausos habent oculos.*"

Can a Woman be Violated during Hypnotic Sleep? The condition of the so-called *hypnotic* or unnatural sleep has given rise to a question connected with the alleged perpetration of rape. There would seem to be no reasonable doubt as to the possibility of such an occurrence; and this is one of the reasons why this method of therapeutics is looked upon with some disfavour, and as one requiring great care on the part of its practitioners.

It is Rape if the Ravisher personates the Husband. It is expressly provided by the Criminal Law Amendment Act, 1885, s. 4, that a person who induces a married woman to permit him to have connection with her by personating her husband is guilty of rape.

In *R.* v. *Morrissey* the accused was sentenced to three years' penal servitude for rape on a married woman in the following circumstances: He entered the woman's room when she was alone in bed and asleep. She awoke as the accused was finishing sexual connection with her, and she believed that it was her husband. The judge said that a man who had connection with a sleeping woman, by personating her husband, *i.e.*, by putting himself in such a position that she might suppose him to be her husband, would be guilty of rape. This case illustrates the difficulties of these cases, which, as a rule, turn on the credibility of the witnesses.

Influence of Hysteria. In a case where a woman, *æt.* 17, alleged that she had not consented to the act, the evidence was that she had been suffering from *hysteria* and was in a fit at the time that the act was perpetrated. The accused was convicted. Cases in which hysteria is pleaded as the cause of unconsciousness should always be treated with suspicion. It is easy for a girl who has given her consent and repented to make an allegation of this kind. A medical witness should ascertain in such cases whether there is any evidence of force or marks of violence on the person or genitals.

Rape on Prostitutes. The law protects a prostitute against involuntary connection just as it protects children and chaste women; but when a charge of rape is made by a prostitute the case should be closely scrutinised. Something more than medical evidence would be required to establish such a charge. The question turns here, as in all cases of rape upon adult women, on whether *consent* had been given previously. This is the point at which the greater number of such cases of alleged rape break down. A medical witness can do no more than to give medical evidence on the question whether or not sexual intercourse has taken place with or without violence. There may be marks of violence about

the pudendum or on the person, and yet the conduct of the woman may have been such as to imply consent on her part; medical proof of intercourse is not legal proof of rape.

Medical Aspects of Rape

The sole object of medical evidence is to determine whether or not connection has taken place.

Medical evidence is commonly required to support a charge of rape, but it is seldom more than corroborative; the facts are, in general, sufficiently apparent from the statement of the prosecutrix. There is, however, one case in which medical evidence is of some importance—namely, when a false accusation is made. In some instances, as in alleged rape on infants and children, the charge may be founded on mistake, but in others it is often wilfully and designedly made, for motives into which it is here unnecessary to inquire. Amos remarked, that for one genuine case of rape tried at Assizes in his time, there were on the average twelve pretended cases. In some few instances these false charges are disproved by medical evidence—in others, medical men may be sometimes the dupes of designing persons. In the majority of cases, the falsehood of the charge is proved by inconsistencies in the statement of the prosecutrix herself. It is stated that in Scotland, where there is a careful preliminary inquiry, false charges of rape are exceedingly rare.

The duty of a medical witness on these occasions will be best understood by considering the subject in relation to females at different ages. On being called to examine a person on whom a rape is alleged to have been committed, a practitioner should notice the precise *time* and date at which he is summoned. This may appear a trivial matter, and one wholly irrelevant to the duties of a medical practitioner; but it is to be observed, that the time at which a surgeon is required to examine a prosecutrix may form a material part of the subsequent inquiry. It will be important to the defence of a person accused, if it can be proved that the female did not take the earliest opportunity to complain; and it may also be the means of defeating an *alibi* falsely set up by an accused person in his defence.

It is rare that cases of rape are tried without medical evidence; occasionally an attempt is made to dispense with it, and the result is generally an acquittal. Juries naturally dislike to convict persons of this serious crime unless the statement of the prosecutrix is corroborated by medical facts and opinions. Medical evidence in cases of rape may be derived from four sources: (1) Marks of violence about the genitals. (2) Marks of violence on the person of the prosecutrix or prisoner. (3) The presence of stains of the spermatic fluid, or of blood on the clothes of the prosecutrix or prisoner. (4) The presence of seminal matter in the vagina. (5) The existence of gonorrhœa or syphilis in one or both. This evidence will vary according to circumstances.

Consent to Examination. In examining the victim of alleged rape it is important to remember that no one, whether coroner, magistrate, judge, or police official, can *compel* a girl to submit to being examined without being guilty of, and running the risk of an action for, indecent assault; hence consent must be obtained before undertaking such an examination (*vide* Vol. I., pp. 40 *et seq.*).

The consent of the victim should be obtained if she is of reasonable age and understanding. If she is not, consent should be obtained from her parent or guardian.

If the circumstances are in any way peculiar, it is advisable to have the consent in writing, and in all cases, without exception, a verbal consent should be given *in the presence of disinterested witnesses.* It is also necessary to state openly that evidence derived from such examination may be used against as well as for the examinee ; this is particularly the case in examining suspected persons.

Having obtained consent, a most thorough examination must be made in a good light and in a position which allows of a good view of the parts.

Previously to the actual inspection of the genitalia it is well to make written notes on the following points.

1. Date and exact hour of the visit.	1. Required principally in reference to lapse of time since the occurrence ; if long delayed, why ?
2. The walk and attitude of the victim, bodily and mental.	2. As to pain on walking ; excited or otherwise ; under the influence of alcohol, etc. Physical and emotional constitution.
3. Who are with her, and their attitude and frame of mind towards her and to the accused.	3. Refers to concocted or genuine tales ; chances of conference as to what to say, etc.
4. The statements of the victim and of her friends separately, inquire especially : (*a*) Age. (*b*) Date, time and place of alleged offence. (*c*) Exact position of the parties, sitting, lying, or standing, etc. (*d*) Did she cry or struggle ? (*e*) Was she menstruating or not at the time ? (*f*) Was she sensible the whole time ?	4. These must be taken down as near as possible verbatim, and will constitute most important evidence either to establish guilt or to free the innocent. She and her friends have probably been to others, or made statements to others, and the general or special agreement of the various statements will be strong proof of their truth or of their falsehood.

The victim should then be undressed, and her clothes examined for—

(*a*) Blood, and its situation. (*b*) Mud and other stains, grass, etc. (*c*) Seminal stains, or suspected seminal stains, on the clothing. (*d*) Tearing, loss of buttons, etc.	All obviously very important, corroborating or contradicting her story.

The physical development of the limbs is noted with reference to her powers of struggling. Bruises and injuries found on the body are described and noted, especially with reference to the possibility of their self-infliction, or to their consonance with her tale. Scratches or bruises may be found about the mouth, arms or legs, inflicted in an attempt to prevent her from crying and struggling or from contact with the ground.

The genitalia, including the breasts, are then carefully examined. It may occasionally happen that the girl is menstruating, or alleged to be so. In clinical practice it is usual at this time to avoid a vaginal examination from motives of decency, but in criminal cases this objection cannot hold; in fact, the establishment of the fact of menstruation may be a most material point.

(*a*) Breasts.	(*a*) In masturbators frequently flabby and large, a point not of great weight, but worth noting.
(*b*) Vulva.	(*b*) Swelling, redness, bruises, tears, tenderness, etc., are all important points that require explanation of their origin.
(*c*) Hymen.	(*c*) Ruptured or intact, age of ruptures.
(*d*) Vagina.	(*d*) Discharges, ulcers, growths in its mucous membrane, etc. A sample of the vaginal contents should be removed for microscopic examination for the presence of spermatozoa and for bacteriological examination. A purulent discharge can hardly arise within twenty-four hours of connection; nor can a venereal sore of any sort appear suddenly.
(*e*) Hairs on pubes.	(*e*) Matting of the hair should be looked for. If there is a foreign substance on the hair, it is necessary to remove some of it for examination for semen, pus, blood, etc.

This represents a complete scheme for the examination of the female. The deductions to be drawn depend very much upon circumstances, of which the most important is the time that has elapsed since the alleged offence. They may vary from completely negative conclusions up to those carrying conviction that violence has been offered to the parts, and (with semen in the vagina) possibly proving that it was a penis that produced them.

If in the power of the medical jurist it is well that the accused should also be examined as soon as possible.

1. Age, size, and strength.	1. The proportion these bear to those of the victim in connection with fear, power to struggle, physical possibility of connection, etc.
2. Scratches and bruises.	2. Evidence of an alleged struggle.

3. Stains on clothing.	3. Seminal or other, possibly capable of innocent explanation.
4. Condition of organs.	
(*a*) Impotency.	(*a*) *Vide pp. 2 et seq.*
(*b*) Smegma on prepuce.	(*b*) Its presence renders recent copulation very improbable.
(*c*) Seminal matters on the organ or in the urethra.	
(*d*) Gonorrhœa.	
(*e*) Venereal sores.	
(*f*) Matting of hairs, etc.	

It must be noted that all these points, even if positive, are quite capable of innocent explanation, for copulation is not illegal, nor is masturbation, but they must be carefully noted for the same purposes as the statements of the victim—*viz.*, to corroborate or contradict the statements made.

Lastly, the site of the alleged offence may be examined.

It now remains to consider with illustrative cases the conditions in which rape occurs.

Rape on Infants and Children up to the age of Sixteen Years

It is in this group of cases that medical evidence is of the greatest importance, because consent cannot be considered.

To the great opprobrium of our civilisation this crime is of frequent occurrence, sometimes as the result of simple lust ; but often from the absurd belief that gonorrhœa, and even syphilis, can be cured by sexual intercourse with a virgin.

The sexual organs should in these cases present **marks of injury** if the crime has been completed, and there has been *any resistance on the part of the child :* for it is impossible to conceive that forcible intercourse should take place without the production of bruising, the effusion of blood, or a laceration of the private parts. Even without reference to manual violence on the part of the assailant, the size of the adult male organ must necessarily cause much local injury in the attempt to enter the vagina of a child. If the violation has taken place within two or three days, the appearances presented by the parts may be as follows :—(1) Inflammation with more or less abrasion of the lining-membrane of the vagina, or the entrance. (2) A muco-purulent discharge from the vagina, of a yellowish or greenish-yellow colour, staining and stiffening the linen worn by the girl ; the mucous membrane of the urethra possibly shares in the inflammation, rendering the discharge of urine painful. (3) In recent cases blood may be oozing from the abraded membrane, or clots of blood may be found deposited in the vulva. (4) The hymen may be entirely destroyed, or (what is more commonly observed) it may present on careful examination one or more lacerations. Owing to the inflamed state of the parts, the proper examination of the hymen is rendered difficult—any attempt to separate the thighs for this purpose causing great pain. For this reason, also, the child walks with difficulty and complains of pain in walking. (5) Last, the vagina may be unnaturally dilated.

In the following case of rape on a young girl, the motive was the superstition alluded to above.

A girl of fourteen was admitted into hospital suffering from constitutional syphilis. She walked with the peculiar gait indicative of pain in the genitals. The hymen was found to have been ruptured some time previously, the two longitudinal folds remaining. There were primary syphilitic ulcers of the external vulva, also condylomata extending from them to the anus. There was a well-marked roseola over the body, and the peculiar state of the throat which accompanies condylomata of the genitals or anus.

She stated that a man, whom she named, had had forcible connection with her the Saturday before the previous Christmas. This very fairly corresponded with her symptoms, remembering that she had received no medical treatment. After obtaining the accused's consent and duly cautioning him as to the result, Lowndes examined him. He found a well-marked indurated chancre, all but healed, and condylomata ani ; the man was in a very dirty and most offensive state. The girl's story was that she remembered the day as the one when her mother went to the pawnbroker's to redeem a lamp she had pledged. This was corroborated by the mother and pawnbroker's assistant. Her parents were both of drunken habits, and it was evident that the accused, who knew their habits well, had watched them go. He sent a young sister of his victim for beer, and another one for apples, then locking the door, threw the girl upon the floor and committed the offence. The girl's story was corroborated in every particular, while an elder sister, who was a prostitute, gave important evidence as to the motive. The accused made overtures to her some time after, and as his diseased condition was well known, she asked him if he was cured. He answered that he had cured himself by connection with a young girl, adding, " You would be astonished if you knew who." He was found guilty.

It has been questioned whether a rape can be perpetrated on children of tender age by an adult man ; and medical witnesses at trials have given conflicting opinions. Some are inclined to regard all such charges as unfounded, and to seek for other medical explanations of the symptoms above described. This practice has been carried to an undue extent, simply because many of these charges have been proved to be false ; but common experience shows that there is too frequently a real foundation for the charge in reference to children, and that a girl is not to be discredited merely because of her tender age.

Sometimes the Violence is Extreme. In *R.* v. *Wood* the peritoneum was penetrated, and the parts about the vagina extensively lacerated.

It is clear that the male organ may produce much physical injury whether the young victim does or does not resist the attempt.

Extensive injuries often are inflicted.

In *R.* v. *Crowley* the prisoner, aged twenty-four, was tried for a criminal assault on his daughter Catherine, who was at the time a little under four years old. Three weeks before the trial the prisoner, who admitted having had nine or ten pints of beer that afternoon, was left alone with the child about 6 o'clock in the evening. On her return, the mother noticed that the child's eyes were blackened and asked the prisoner what he had been doing. He made a remark that it was not her eyes but that she was " bleeding below." The mother then saw that the child was naked, and that her privates were torn and bleeding.

A bucket containing bloody water was found in the house.

The child was very well developed for her age. There was ecchymosis about each eye, and several slight bruises on the body.

The Perineum. The labia majora were swollen and bruised, the right especially so. The fourchette had been torn across, as well as the posterior wall of the vagina, for a depth of rather over a quarter of an inch. Extending backwards from this there was a deep laceration which came up to the anterior wall of the rectum (this last structure was intact) ; the anterior part of the external sphincter, *i.e.*, that part between the anal orifice and the central tendon of the perineum, had been torn. On admission to hospital there was only slight oozing of blood.

The child was anæsthetised ; and the laceration, after careful cleansing, was closed with four deep sutures of silk-worm gut. The prisoner expressed a wish to be examined by a doctor, and was examined by the police surgeon, who first obtained the prisoner's consent, in the presence of witnesses, and cautioned him as to what the result of the examination might be. He consented and undressed. There were no marks of violence on his person nor any stains of blood, semen, etc. His pudenda were unusually clean for a man of his employment, and cleaner than the rest of his body. He had had time and opportunity to wash himself. He was charged with having carnal knowledge of his daughter, and in reply said, " I know nothing whatever about it." The apron in which the child had been wrapped when she was taken to the hospital contained blood and spermatozoa on one spot, and blood only on the other. There was nothing found elsewhere. The clothes had been washed and left wet, until they smelled horribly and swarmed with microscopic organisms, and the water in the bucket had been thrown away.

The prisoner's story was that he was washing the child in the bucket (an ordinary wooden one) when she slipped from his hands and fell astraddle of the bucket, the injuries being caused in this way. The medical evidence was that it was impossible that the injuries could have been caused in the manner described by the prisoner. The child told her mother that " daddy had done bad in the bucket," but this could not be given in evidence. The mother was a willing witness against her husband and her evidence was corroborated by a neighbour. The jury wished to know why no marks of violence were found on the prisoner, and a medical witness stated that the male organ, being pressed against soft parts, would not show marks of violence, and that the injury to the child could not have been caused in the manner described by the prisoner, who was sentenced to ten years' penal servitude.

A rape committed by an adult on a child eight years old terminated fatally from peritonitis, as a result of the violence, six days after the assault. The child stated that the accused had had forcible connection with her, causing much pain and loss of blood. There were no marks of violence (bruises ?) externally, but the orifice of the vagina was lacerated in its entire circumference, and the perineum was nearly torn through. It was found, on inspection, that the orifice, as well as the whole of the vagina, was in a state of gangrene, and that its posterior wall had been lacerated at its line of junction with the uterus to the extent of an inch. The labia and clitoris had not undergone any change. The prisoner subsequently confessed his guilt.

In another case the victim was about six years of age, and very intelligent. From her description of the assault, it appears that she fainted, probably owing to the severity of the pain. When examined, it was found that the vagina was ruptured in various directions. One laceration extended from the lower part downwards, dividing the recto-vaginal septum and perineum down to the verge of the anus. There was a lacerated opening in the coats of the rectum ; the orifice of the vagina was lacerated upwards as well as laterally ; the parts were raw, swollen, and very tender. When the child was first seen there was blood on the limbs and clothes ; she recovered from these serious injuries in about two months.

In a case of alleged rape on an infant only eleven months old, the violence done to the genitals proved fatal. During the march of a regiment, the prisoner, a soldier, who was with the sick car, took the child from its mother to carry it some way for her. The child was quite well when he took it : he walked on quickly, and was out of the mother's sight in half an hour. When she came up, he had the child standing on the grass facing him, and he was bent over it : with one hand he held the child's petticoats up, and the other was covered with blood. He told the mother that the child was ill and passing blood. The mother rolled it in her shawl, and carried it to an apothecary ; but no examination was then made, and it was not until the next morning that, in washing the child, the marks of violence were seen. This was the substance of the mother's evidence, which was uncontradicted at the trial. A surgeon examined the child twenty hours after the alleged outrage : it was then in a state of collapse, and it died in a few hours. All the external parts of generation were found in a torn state, and violently inflamed ; the perineum was torn nearly through ; the nymphæ and the mucous lining of the labia and clitoris were likewise lacerated, so that the whole presented the appearance of a large lacerated wound in a state of severe inflammation. After

death, besides the above-mentioned appearances, the vagina was found greatly dilated and torn from its attachment to the neck of the womb posteriorly, making a large opening into the cavity of the abdomen, in which a quantity of bloody serum was effused. Wilde, on making inquiry into the particulars of this case, ascertained that there was no proof of the actual perpetration of rape. The severe injuries to the genital organs which led to death were produced, it was alleged, by the fingers—the man being at the time partially intoxicated.

A girl, seven years old, was admitted into Guy's Hospital suffering from injuries resulting from rape by a boy under seventeen years of age. About half an hour had elapsed when she was examined. There was found a complete destruction of the hymen, with a laceration of about one-eighth of an inch extending into the perineum. There had been profuse bleeding, as the clothes were saturated with blood. There was then no complaint of pain, and there were no scratches or marks of violence on any part of the body. There was no discharge of a purulent kind. The child was of a scrofulous habit ; but she was not suffering from vaginitis, and appeared in other respects perfectly healthy. Forty-eight hours after the occurrence the bleeding had ceased, and the extent of the lacerations was very perceptible. There was no discharge of any kind from the vagina, and no inflamed or swollen condition of the parts. The boy was examined about an hour after the perpetration of the rape, and although he had been under strict custody, and had had no opportunity of changing his clothes, there was *no blood* found about his private parts, or on his clothing. It is probable, as the boy was interrupted in the act by the screaming of the girl, that he suddenly withdrew after having caused the laceration, and that the bleeding was an after-effect of oozing from the ruptured vessels.

This **absence of blood on the accused** is important, because the main facts of the case were indisputable ; such absence of blood might be construed into evidence of innocence. It is clear from the above case and the two following that such an inference is quite unjustifiable.

Sawyer met with a case in which a rape was committed on a girl *æt.* 5. There was a bruised and swollen state of the genitals ; the hymen was not ruptured, and there was no laceration of the parts. In spite of this, a large amount of blood had been lost. This bleeding, he considers, took place from the hymen, which was in a highly congested state. The man who had perpetrated the crime was examined soon afterwards, but no appearance of blood was found on his organs ; there were a few stains only on the front of his clothing.[1] A case occurred to Sells, in 1863, in which he found on examining the person of a girl said to have been violated, laceration of the hymen, a clot of blood recently effused lying on the vulva, and the thighs of the child smeared with blood, quite fresh ; there was also blood on the sheets of the child's bed. The next morning he examined the accused, but he could find no trace of blood upon him or on the clothing which he wore at the time of the alleged assault. In this case, as there was a failure of identification, the accused was discharged.

If no Marks of Violence are Found. When there are no marks of violence or physical injury about the pudendum of a child, whether because none originally existed or they existed and had disappeared in the course of time, a medical witness must leave the proof of rape to others. He can only answer questions of possibility, or probability, according to the special facts proved. It is, however, in all cases his duty to be guarded in giving an opinion that a rape has been perpetrated, when there is a total absence of marks of violence on the genitals. Rape may be committed without necessarily producing such marks on a child, in which case proof of the crime will not depend on *medical evidence* only. The absence of marks of violence on the genitals of the child, when an early examination has been made, is strong evidence that rape has *not* been committed. A false charge might easily be made and

[1] *New Orleans Med. Gaz.*, 1858, p. 283.

sustained, if medical opinions were hastily given on the statements of the mother and the child in the absence of physical appearance to corroborate the accusation.

Violence Found, but its Origin possibly Doubtful. In cases where the examination of the child took place within a very short time of the alleged offence, it should be comparatively easy for a medical practitioner to ascertain whether or not violence has been inflicted by penis, or finger, or otherwise ; but marks of violence must not always be hastily assumed to furnish proof of rape ; for cases are recorded in which injuries have been purposely produced on young children as a ground for bringing false charges with the view of extorting money. The proof or disproof of facts of this kind must rest more upon general than upon medical evidence, unless the injuries obviously indicate the use of some weapon or instrument.

Chevers states, on the authority of a missionary well acquainted with the habits of the natives of Calcutta, that mechanical means, especially by the use of the fruit of the plantain, are commonly employed, even by the parents of immature girls, to render them *aptæ viri*. In one instance a man was convicted of rape who, according to the evidence, had previously used a small stick—*ad deobstruendam viam*. This led to effusion of blood, but to no permanent injury. It is scarcely credible that human mothers should resort to such practices, nevertheless the facts are too well accredited to be denied. Casper examined a girl only ten years of age, whose vagina had been dilated by the mother, at first with two fingers, afterwards with four, and finally by means of a long stone introduced into it, in order to fit her for intercourse with men. The hymen was not destroyed, but there were lacerations in it ; the mucous membrane was reddened and painful to the touch, and there was a mucous discharge from it.[1] This shows that medical evidence can do no more than show that a girl with such appearances about her sexual organs has suffered from some violence applied to the part, but whether by the male organ, or any other physical means, it would be impossible to say.[2]

If, moreover, there has been considerable delay before inspection, inflammation, followed by sloughing or mortification, may have set in, and such may destroy life, especially in children of an unhealthy habit.

Noma Vulvæ. Care should be taken that the symptoms of a malignant form of disease (noma, a form of destructive ulceration), to which female children are sometimes subject, are not mistaken for criminal violence. In the following case of *noma pudendi* no charge of rape was made against any person, but the facts may serve to show in what circumstances such a charge might be made.

A girl, *æt.* 5, died, as it was suspected, from the effects of poison. There was a congested state of the stomach, but no poison was found. The genital organs externally, and the skin around and beyond the anus, were intensely inflamed, swollen, and ulcerated, and in an approaching state of gangrene or sloughing. The hymen was destroyed posteriorly, and the lining-membrane of the vagina and uterus was much inflamed, of a dark purple colour, with softening and disorganisation of substance. The upper inguinal glands were enlarged on both sides. The child was in a neglected and dirty state. The mother attributed this

[1] "Gerichtl. Med.," vol. 2, p. 162.
[2] 3 Casper, E. Tr. 318.

diseased condition of the genitals to a fall which the girl had met with a fortnight before. There was no ground to believe that anyone had had connection with the deceased.

A girl, aged seventeen, was violated by several men in succession ; she then became insensible, and was unable to state how often the act had been perpetrated. When examined the next day the genitals were bloody, inflamed, and painful ; the hymen was ruptured, the fourchette torn, and the labia and perineum presented a dusky appearance of inflammation. In spite of treatment ulceration followed, and the clitoris, nymphæ, perineum, labia, and mons veneris sloughed away, leaving the pubis exposed. After a long illness the ulcer healed, and the girl left the infirmary. At no period were there symptoms of syphilis. Such a state of the parts, obviously a result of violence, might have been erroneously ascribed to *noma* or malignant ulceration or mortification of the genitals, as is observed in some eruptive fevers.

Evidence from Vaginal Discharges. The existence of a purulent discharge from the vagina, as a result of vaginitis or inflammation of the vagina, has been erroneously adduced as a sign of rape in young children. The parents, or other ignorant persons who examine the child, often look upon this as a positive proof of intercourse ; and perhaps lay a charge against an innocent person, who may have been observed to take particular notice of the child. Some cases are reported by which it would appear that men have thus narrowly escaped conviction for a crime which had not been perpetrated.

A purulent discharge with aphthous ulceration is occasionally a result of simple, *i.e.*, non-venereal vaginitis (inflammation of the vagina) in young children. It may arise from local causes of irritation such as worms or uncleanly habits. It is met with in girls up to six or seven years of age ; and children thus affected have been tutored to lay imputations against innocent persons for the purpose of extorting money.

Such discharges of a simple nature may occur, but accurate bacteriological examination of pus from cases has tended to show that a large proportion, something like 75 per cent., of them are gonorrhœal, a position which is easily understood since we know how widely spread the gonococcus is, and how numerous are the opportunities for its transfer to the pudenda of small children.

Numerous instances of such transference from flannels and sponges, and various other articles, have been quoted. Ryan long ago traced the origin of a discharge in two children to their being washed in a vessel of water with a sponge used by a young woman infected with gonorrhœa.

If a discharge is present, it should be examined microscopically, and suitable cultures prepared and examined for the gonococcus.

Inasmuch as most of such discharges are gonorrhœal, it remains to discuss the bearing of the fact on the question of the alleged offence, and the nature of the medical evidence which can be given either for or against the accused.

Time is the first important factor. When gonorrhœa is acquired in the usual way by sexual connection there is always an interval of from two to four days (occasionally it may be nearly a week) between infection and the appearance of a discharge with marked inflammatory redness of the mucous membrane. When syphilis is similarly acquired the interval between connection and the appearance of a chancre is very much longer, usually about three or four weeks ; with other syphilitic manifestations (rash, condylomata, etc.) the interval is still longer (six weeks to three months). When other venereal troubles are thus caused

the interval is of less certain duration, but commonly from two days to a week elapses before a discharge is seen with evident ulceration.

The deductions to be made from these facts are self-evident, but they vary with each set of circumstances. It is most important to remember that a discharge cannot appear within twenty-four hours of the offence if the offence was the cause of the discharge. If there is a discharge, and if the offence is alleged to have occurred some days previous to examination, then the proof of cause and effect will have to depend upon other factors. The examination of stains on the underlinen may show that a gonorrhœal discharge was present in the female at the time of the offence.

The next question which arises is whether, assuming that one of the two parties is suffering from a venereal infection and the other is not, it is certain that the disease will be communicated to the other party ? The probability of infection is very great, but facts would seem to prove conclusively that infection is by no means certain.

A man may be *apparently* free from disease and yet have in his urethra gonococci which in him are incapable of causing a discharge, but which transplanted into the vagina, may there cause a severe attack of gonorrhœa with profuse discharge ; this fact has been proved over and over again by clinical observation as well as by bacteriological research.

If a vaginal discharge is present the medical man must make minute inquiries as to the time when it was first observed and compare this with the time of the alleged offence in order to ascertain whether it is possible for the relationship of cause and effect to be established. This relationship is much easier to establish or refute in the case of syphilis and of gonorrhœa than of other venereal troubles. If this possibility be established he should examine the accused, if there be such a person, and see if the same chain of evidence is complete.

Hamilton relates a case in which syphilis was communicated to a girl, *æt.* 6, by a boy aged 19. The accused was found to have numerous sores around the orifice of the prepuce, and on examining the little girl there were chancrous excoriations on the inside of the labia. Other syphilitic symptoms manifested themselves. The prisoner was convicted.

It is in cases such as these that the examination of the accused affords such excellent evidence, either confirmatory of his guilt or strongly suggesting his innocence.

Gonorrhœa is a disease of very uncertain duration. It may continue for weeks, months, or even years ; it is therefore very unwise for a medical man to express an opinion as to its duration.

In conclusion we may observe that discharges from the vagina should not be admitted as furnishing corroborative evidence of rape, except,—first, when the accused party is suffering from a gonorrhœal discharge ; second, when the date of its appearance in a child is from the third to the eighth day after the alleged intercourse ; and third, when it has been satisfactorily established that the child had not suffered from any such discharge previously to the assault. It may be said, however, that all these conditions may exist, and yet the accused be innocent; for a child may, either through mistake or design, accuse an innocent person. This, however, removes the case entirely from the hands of a medical jurist.

Evidence from Violence other than that to the Pudendal Regions. Marks of violence on the *body* of a young child are seldom met with, because no resistance is commonly made by mere children. Bruises or contusion may, however, be occasionally found on the legs.

The older the victim, the greater is the possibility that such marks will be found on the body, and the more the importance to be attached to them when found, or at least to the explanation of how they were produced.

RAPE ON GIRLS OVER SIXTEEN AND ON ADULT WOMEN

In these cases medical evidence is of much less value than in the previous classes of cases, for though it may still be able to prove the fact of connection having taken place it can have very little influence upon the question of consent, the absence of which constitutes the essential element of rape. We must, however, consider the evidence which it may be possible to give.

Evidence from Violence to Parts other than Pudendal. Girls who have passed the age of sixteen, and adult women, are considered to be capable of offering some resistance to the perpetration of the crime. Therefore in a true charge we should expect to find not only marks of violence about the pudendum, but also injuries of greater or less extent upon the body and limbs.

With respect to such marks of violence on the person, the exact form, position, and extent of these should be noticed; because a false accusation of rape may sometimes be detected by the marks of violence being in a situation in which they could be produced only with great difficulty by an assailant. When bruises are found, the presence or absence of the usual zones of colour may occasionally (*vide* Vol. I., "Bruises") throw light upon the time at which the alleged assault was committed. As these marks of violence on the person are not likely to have been produced with the concurrence of the girl, they are considered to furnish some proof of the intercourse having been against her will. Bruises upon the arms particularly may be considered to be reasonable evidence of a struggle; impressions of finger nails, too, are suggestive. Bruises or scratches about the inner side of the thighs and knees may be inflicted during attempts to abduct the legs forcibly.

Strong corroborative evidence of a struggle might be obtained from an examination of the accused for similar marks of bruises or scratches about the arms or face, and possibly even about his penis, though this is much less likely.

Evidence from Violence to the Pudenda. In considering this point we must draw a distinction between a virgin and a married woman who is in the habit of having sexual connection. In the latter it is extremely unlikely that marks of violence to the private parts will be found, though they must be looked for, because it is commonly assumed that rape is associated with greater violence than sexual connection with consent. In a virgin the physical appearances of rape about the genital organs may be found, whether the connection has been voluntary or involuntary. Thus a recent rupture of the hymen, laceration or bruising of the vagina with effusion of blood, swelling and inflammation of the

vulva, and stains of blood upon the person, dress, or *locus* may be met with in either case. The question of consent in these cases is of great importance. It is generally alleged as a defence, and a medical man will find himself compelled to answer this question : Are the marks of violence found on the genital organs no more than you would expect to find in a girl who had really given consent ?

A man with a wooden leg (his left leg having been amputated at the thigh) was charged with rape on a girl, *æt.* 15. She was examined soon after the violence, and the labia were found very much swollen, bruised, and inflamed. In addition to these appearances on the genital organs, there were the marks of bruises over the right chest, breast, and shoulder. These latter certainly suggested that connection had been violent without consent, but the man alleged that the girl gave her consent, whereupon the following question arose :—Could such appearances about the labia have been produced by connection with consent ?

The condition of the genital organs and the marks of violence on the body in this case were adverse to the theory of consent ; but in expressing an opinion in such circumstances it must be remembered that, from the difference in the size of the organs of an adult male and a girl of fifteen years of age, it is hardly probable that intercourse with consent could take place without causing subsequent swelling and inflammation of the labia and vagina. In making an examination, the greatest care should be taken by the practitioner to fix, at the time of examination, a probable date for the marks of injury to the genitals or other parts of the body, as it is by the aid of such observations that the truth or falsity of a charge may be sometimes clearly established.

It is probable that in the case of adult virgins, if the charge were well founded, the hymen would be ruptured, as the intercourse is always presumed to be violent : but there might be some degree of penetration without this being a necessary result, especially if the membrane were small, or placed far up. At any rate, a girl may sustain all the injury, morally and physically, which the perpetration of the crime can possibly bring upon her, whatever may have been the degree of penetration ; and for this, reason it is laid down by the law of England that the crime consists in the mere proof of penetration. Girls of tender age are sometimes violated by boys ; the amount of physical injury inflicted in such cases is usually less than when the assailant is an adult.

When a woman has already been in the habit of sexual intercourse, there is much less injury done to the genital organs. The hymen will, in these cases, be found destroyed and the vulva dilated. Nevertheless, as the intercourse is presumed to be against the consent of the woman, it is most likely that when there has been a proper resistance, some injury may be apparent on the pudendum ; and there will be also, probably, marks of violence on the body and limbs. These cases are generally determined without medical evidence by the deposition of the woman, corroborated, as it should always be, by circumstances. This statement regarding the presence of *marks of violence* on the pudendum of a married woman, on whom a rape is alleged to have been committed, requires some qualification. In two cases of rape on married women, in which the crime was completed in spite of the resistance of the women, there were no marks of violence on the genital organs.

In one case, while an accomplice held the head of the woman with her face downwards, between his thighs, the prisoner had forcible intercourse with her from behind—her thighs having been first widely separated. In another case an accomplice held the woman down on a bed by her neck, while the prisoner separated her thighs, and thus had intercourse with her. She was examined nine hours afterwards by an experienced surgeon, and he found no mark or trace of violence on or anywhere near her pudendum. There were bruises on her arms, neck, and legs, where she had been forcibly held down.

In each of these cases, it will be seen that the woman had to struggle with more than one assailant; and there can be no doubt that if a married woman is rendered powerless by the combined efforts of two or more men, or if she is rendered insensible by intoxicating drinks or narcotic vapours, a rape may be perpetrated without any injury whatever to the genital organs. A separation of the thighs in a married woman will cause such a dilation of the parts, as to render it easy for the male organ to penetrate the vagina without leaving any traces of violence.

On the other hand, the vagina may be the seat of violence, and no marks to indicate a struggle or the application of force may be found on the body.

A woman was knocked down, her clothes were pulled over her face, and a rape was perpetrated by the assailant. In the position in which she was held, with her arms and hands covered over, she was half suffocated, and unable to offer any effectual resistance. She was examined on the evening of the day of the assault. No marks of violence were found on her body, but the mucous membrane of the vagina at its commencement was contused, and in some portions lacerated; and blood was oozing from these parts.

The statement of the woman was consistent with the fact that there were no marks of violence on her body. There was no reason to suppose that the injury to the vagina had been caused in any way other than by a criminal assault.

Evidence from the presence of Spermatozoa. Proof of emission is not required by the law, and therefore evidence of the presence of spermatozoa is not indispensable. Nevertheless, it is necessary to examine any spots and stains on the clothing of the prosecutrix or of the accused for the presence of semen.

The vagina of the victim and her pubic hair should always be examined also. The presence of spermatozoa in the vagina is conclusive proof of connection, but not of rape; their absence is no proof that connection has not taken place, for they may have been removed by washing or by discharges. The presence of mobile spermatozoa in the vagina indicates that connection was recent, *i.e.* within a few hours of the time of examination.

We have stated that a rape may be legally completed without emission; it is quite possible, however, that there might be marks of emission without penetration.

In a case of rape perpetrated on a child, some woollen fibres of a blue and red colour were found, in addition to blood-corpuscles. This observation aided in fixing the identity of the assailant, since it was proved that the man wore a red flannel shirt over a bluish-grey woollen shirt.

A medical witness must be prepared to consider the precise value of evidence furnished by the microscope in the examination of stains on the dress of a man accused of rape. A shirt may present stains of

blood, urine, mucus, or gonorrhœal discharge, some of which, but for the microscope, might be mistaken for spermatic stains. Although it is true that the microscope enables an examiner to affirm that the stains have been caused by the spermatic secretion, this does not prove that a rape has been committed, or even that intercourse has been had with a woman. Such stains may arise from natural discharge, or from disease (spermatorrhœa), and therefore of themselves they afford no proof of intercourse. If, from other circumstances in the case, it should be clearly and satisfactorily proved that there has been intercourse, then the presence of blood mixed with the spermatic stains might, in certain cases, justify an opinion that violence had been used. The discovery of spermatic stains on the dress of a woman furnishes stronger evidence of intercourse, attempted or perpetrated, than their discovery on the dress of a man; but, admitting that intercourse is thus proved, it may still have taken place with the consent of the woman. These stains, when found on the clothes of girls and infants, afford a strong corroborative proof of the perpetration of or of the attempt to perpetrate the crime.

Evidence from Vaginal Discharges. If discharges were confined to those of a *recent* gonorrhœal origin, a very large section of Gynæcology would remain unwritten, and for our present purpose it is only recent gonorrhœa that can be taken into consideration.

That vaginitis with a muco-purulent discharge has been observed to arise during febrile complaints, scarlatina, measles, etc., is well recognised. Moreover, diphtheria of the vagina in children and adults may lead to destruction of the hymen, and even more serious trouble (*vide* "Impotency"). Therefore when a woman alleges that a purulent discharge has arisen as a result of rape, the case requires to be investigated with great care, especially in regard to its nature (bacteriologically defined) and its duration.

It is possible that a woman suffering from leucorrhœa may charge a man with the crime of rape, and affirm that this discharge had arisen from the act of the man. An inflamed and partially ulcerated state of the lining-membrane of the vulva may apparently give support to the accusation. It is impossible without a bacteriological examination to distinguish such discharges from those of gonorrhœa, for a leucorrhœal discharge may resemble that of gonorrhœa. Such discharges, commencing before, but continuing and sometimes becoming aggravated after marriage, have given rise to unfounded suspicions of infection from venereal disease imparted by the husband, and have thus led to suits of divorce (*vide* "Divorce").

A young married woman suffered from a discharge which was pronounced by a medical man to be gonorrhœal. This led to proceedings for divorce. A further examination by other medical practitioners, with a complete history of the symptoms from which she had suffered, led to the conclusion that she was suffering from severe leucorrhœa when she was married, and that this was followed by granular vaginitis which accounted for the muco-purulent discharge.

Bacteriological examination is the only safe means of distinguishing gonorrhœal or other venereal discharges from ordinary purulent discharges, the clinical appearances, history and course of a vaginal discharge being

very uncertain guides in legal medicine. We have already (*vide* p. 87) given the principles on which medical evidence must be founded, the adult vagina differing in this respect not at all from that of the child.

Loss of Physical Evidence

The indications of rape, however well marked they may be in the first instance, soon disappear or become obscure, especially in those women who have been already habituated to sexual intercourse. After two, three, or four days, unless there has been an unusual degree of violence, no traces of the crime may be found about the genital organs. In the case of an adult married woman the appearances of injury discovered in and about the vagina had begun to heal in less than forty-eight hours ; but in another case examined on the ninth day, the lining membrane of the vagina was still reddened, and the parts were painful. In this case the hymen was completely torn through. In married women, or in those accustomed to sexual intercourse, no inference can be drawn from a dilated state of the vagina. In unmarried women, and in children where there has been much violence, the signs of rape may persist and be apparent for a week or longer. Assuming that they are not found at the time of examination, it may be necessary to consider whether there has been time for them to disappear since the alleged perpetration of the offence ; but in such cases it is rarely in a medical witness's power to express an affirmative opinion as to the perpetration of the crime.

A man, *æt.* 37, committed a rape on a girl only eight years of age ; he was seen in the act, and defended himself on the plea of drunkenness. The girl was examined by a medical man on the day following. The labia were reddened, and there was injection of the membrane at the entrance of the vagina, which was very sensitive. As an illustration of the rapidity with which the marks of rape disappear in young children, when not accompanied by great physical injury, this girl was carefully examined *eleven days* after the assault, when the sexual organs were in their natural state, and there was not the least appearance of local injury.

Medical practitioners are not always sufficiently careful in the inferences which they draw from an examination of children made long after the alleged rape took place. They may be deceived by a plausible and apparently consistent story, and may imagine upon examining the sexual organs of a girl many weeks after the alleged perpetration of the crime that proofs of rape exist. The delay in having the examination made, unless satisfactorily explained, should always be regarded as a suspicious circumstance.

A man was tried on a charge of rape on a girl a little over seven years of age. About *six weeks* had elapsed before the girl was seen and examined by the medical man, who was the only witness for the prosecution ; and after this long period he was prepared to swear at the trial that a rape had been perpetrated on the child. The child was the first witness called ; and under cross-examination, she admitted that all that she had previously stated before the magistrates regarding the prisoner was untrue. Her evidence so clearly established the innocence of the man, that the case broke down, and he was at once acquitted.

But for the medical evidence against him, this man could not have been committed for trial on the charge ; and it is therefore desirable to consider the medical facts and opinions on which he was committed. The medical man came to the conclusion that the girl had been violated

six weeks before he examined her. In his opinion there had been penetration ; the vagina was unnaturally dilated ; there was a discharge from it, and an abrasion on the left side ; the mucous membrane was generally inflamed. "Such appearances might have existed as the result of violence perpetrated on them three months previously. He had frequently examined the girl since, and his conclusion from the first examination had been confirmed. He thought the appearances could not be the result of any accident or disease ; it was not impossible but improbable that they might be so." From what has been already stated on the medical proofs of rape, it will be obvious—(*a*) That in this case there was no evidence of penetration by the male organ, and that the appearances, after six weeks had elapsed did not in any way justify such an opinion from an examination then made. (*b*) That the discharge, the abrasion, and the inflammation of the vagina were all explicable on other grounds, and did not prove that a rape had been committed on the girl at the date assigned. It is highly probable that this child was suffering under that kind of inflammation and purulent discharge from the genital organs which has been elsewhere described as a fertile source of medical errors (*ante*, pp. 87 *et seq.*) ; but whether this be admitted or not, there was not the slightest proof, from the facts, that this girl had ever been violated, even assuming that her own evidence had not shown that the medical man had come to a wrong conclusion from the data before him. Dilatation of the vagina, if really present, could not have been the result of only one attempted intercourse with a child of such tender years, six weeks before the date of examination. This is a most flagrant example of biased and improper medical evidence.

When there has been great laceration of the sexual organs, then certain appearances in the form of cicatrices may remain ; but in all cases great caution should be observed in giving an opinion that rape has been perpetrated, from an examination made even two or three weeks after the alleged commission of the offence. Marks of violence on the person can never establish a rape ; they merely indicate that the crime may have been attempted.

False Charges of Rape

False charges of rape are sometimes brought by girls at the age of puberty. The falsity of the charge may, however, be generally elicited by a careful examination of the girl.

In order to exculpate an unjustly accused man, great circumspection and very close examination of the evidence may be necessary. The medical evidence can rarely be more than merely negative ; it is practically impossible from such evidence alone to say that there may not have been such a degree of penetration as constitutes rape in law. The crime is so rightly detested that few men are prepared to face a trial even for attempted rape or indecent assault, especially on a little girl. When a girl over sixteen or a woman is in question, juries are very inclined to take the view that "there cannot be smoke without fire."

In a case of this kind, a woman charged a youth with having committed a rape upon her infant child.

On examination the sexual organs were found uninjured; and on inspecting the marks of blood on the clothes of the child, it was observed that the stains had been produced on the *outside*, and bore the appearance of smearing; the whole fibre of the stuff had not even been completely penetrated by the liquid. These facts established the falsehood of the charge.

In a case involving a false charge of rape, one of the witnesses (an accomplice), said that she had purchased some blood and handed it to the woman who made the charge, and that she saw her smear it over her person and on some sheets on which it was alleged the rape was perpetrated. The woman who made this false charge, and her husband, were convicted of conspiracy.

The following case of alleged rape is interesting *apropos* of a medical man's duty in investigating such cases :—

A servant girl of good character rushed home one night, knocked excitedly at the door, and when her mother opened it, fell fainting into her arms. On coming to, she told the following extraordinary story : She had been out walking with another girl, and had passed a man whom she could not describe. She and the other girl had separated at the end of the road, and she had then come back along in the direction of where the man had been. When she reached a lonely part of the road she had been thrown down by him on her back, hurting it against a stone. A white scarf had been placed over her mouth to prevent her screaming, and the man, holding down her arms, had had forcible intercourse with her. In less than half an hour the police had searched the road, but no scarf had been found. Upon examination there was evidence on her dress and hat that she had been on the ground. A red mark was seen in about the middle of her back, where she complained of pain, but, upon examination, there was no evidence of the alleged rape ; there were no stains on the clothes, no marks of violence, not even a scratch on her limbs ; no rupture of the hymen, and no sign of any irritation about the genitals. The girl had probably been thrown down, and in her fright, had imagined the rest.

Pregnancy following Rape

Conception may and often does follow rape ; for conception does not depend on the consciousness or volition of a female. If the state of the uterine organs be in a favourable condition, impregnation may take place as readily as if the intercourse were voluntary : and penetration is not necessary for impregnation.[1] Insemination by means of a syringe has frequently led to a successful impregnation. The extrusion of an ovum does not depend on the will of a woman, but is a periodical condition ; the action of the spermatozoa on this ovum is as much removed from the will of the woman as it is from that of the man.

Evidence of Violation in the Dead

The body of a child or woman is found dead, and a medical man may be required to determine whether her person has or has not been violated before death. There is here some difficulty, because there may be no statement made by the deceased. The witness can seldom do more than express a conjectural opinion from the discovery of marks of violence on the person and about the genital organs. Even if spermatozoa were detected in the liquid mucus of the vagina, or on the dress of a female, this would merely prove that there had been intercourse ; whether violent or not would depend on the medical and circumstantial evidence.

[1] See *B.M.J.*, February 16th, 1924, for case of pregnancy in a girl with a tight unruptured hymen.

In a case of murder tried at Edinburgh, the first question to be determined in the dead body was, whether a rape had been committed. The examination of the stains on the clothing was conclusive, when taken in conjunction with the other evidence. The jury convicted the man of rape, but acquitted him of the murder, although the proof of the latter crime was clearer than that of the rape.

In *R.* v. *Kerr*, the evidence of rape consisted of two lacerations of the victim's vagina (which, in an elderly woman who had borne two children, indicated violence), an excoriation on the abdomen ; and blood on the external genitals. The accused had an abrasion on his cheek, vomited matters on the back of his coat resembling exactly that which was exuding from the mouth of the deceased, while on the knees of his trousers were found embedded in mud (resembling that found in the locality where the deceased's body was found), some red woollen hairs resembling that of a petticoat worn by the deceased woman. The cause of the woman's death was suffocation from the impaction of vomited matters in the larynx, but as this was the result of an unlawful act—*i.e.*, rape—it became murder. Two men who knew him intimately saw the accused coming from the spot where the deceased's body was found and within a short distance from it. He and the deceased were walking in opposite directions at such times and places as would bring them to that spot shortly before the time he was met coming away, and when arrested some hours afterwards, his clothes were found wet and muddy. He was found guilty.

Rape by Females on Males

Although there appears to be no specific crime known to English law as rape by females on males, the Court of Criminal Appeal has held in *R.* v. *Hare*[1] that a woman can be convicted of an indecent assault on a boy under sect. 62 of the Offences Against the Person Act, 1861, and also that a woman can be convicted of an indecent assault on another female under sect. 52 of the Act. The following cases occurred in France :

A girl, aged eighteen, was found guilty of an act of indecency, with violence, on the person of a boy under the age of fifteen years. In another case, a girl, aged eighteen, was charged with rape on two children, the one eleven and the other thirteen years of age. The girl enticed the two boys into a field, and had forcible connection with them. This female was proved to have had an unnatural contraction of the vagina, which prevented intercourse with adult males. She was found to be suffering from syphilitic disease, and the proof of her offence was completed by the disease having been communicated to the two boys. She was convicted.

By the Penal Code of France, it is a crime in either sex to attempt intercourse with the other, whether with or without violence, when the child is under eleven years of age. There is no doubt that this offence is committed in England. It is not unusual to find, in the wards of hospitals, mere boys affected with venereal disease. In some instances this may be due to precocious puberty ; but in others it can be ascribed only to that unnatural connection of adult women with boys which is punished as a crime in the other sex. Medical evidence in such cases is usually inconclusive, for there is little chance of any observable effect on the boy. The definite proof of transmission of gonorrhœa or syphilis from the woman to the child provides evidence of a confirmatory nature if it can be proved that the woman is suffering from the disease and that the boy was free from it before the alleged assault, and that the onset of the disease took place within the incubation period.

Case in which Matters associated with Spermatozoa proved Important

Starch, it is well known, is rendered blue by iodine. As stained articles of dress sent for examination may contain starch used for washing

[1] [1934] 1 K.B., 354. The boy (æt. 12) had been infected with a venereal disease.

purposes, a bluish colour may be observed on the addition of iodine, forming a strong contrast with those bodies which are turned of a yellow colour by iodine.

In a case of alleged rape, spermatic stains on the clothing of a little girl showed on the application of iodine, distinct unbroken granules of wheat-starch and potato-starch of a blue colour. These could not have been derived from the starch used in washing, as the granular structure is there destroyed, and, further, the granules were found only in the spermatic stains, and not on other parts of the linen. It was ascertained that the accused used flour in his business, and that there was an open sack of flour at the foot of the bed on which he had committed a rape; some of this had been spilled in the struggle, and had adhered to the stains on his shirt. The flour in the sack was a mixture of wheaten flour and potato-starch. This discovery furnished strong evidence against the accused.

Tests for Seminal Stains

As in the case of blood, the examination for seminal stains may be said to consist of several distinct processes.

1. Inspection by ultra-violet light.
2. Inspection with naked eye and lens.
3. Microchemical test.
4. Microscopical examination.
5. Biological tests.

1. If the garments are examined under filtered ultra-violet light areas of staining by seminal discharges show a bluish fluorescence. This fluorescence is not specific, but it may enable the observer to detect suspicious areas which might otherwise be missed. Such areas are marked for further examination.

2. *Inspection with Naked Eye and Lens.* In nearly all cases the stained articles are presented for examination in the dried state. It is but rarely that a case occurs in which a medical jurist is required to examine a liquid preparation of spermatozoa, though if a girl is brought to him *shortly* after an alleged rape, he may try to obtain living spermatozoa from the vagina. The appearance presented by such a stain on undyed linen or cotton articles, on woollen or other rough articles, and even on dyed smooth fabrics, is like that produced by a dilute solution of albumen. It is of a faintly yellow colour, the stuff is stiffened slightly, and has a somewhat translucent appearance, especially at the edges.

3. *The Preparation of Material and Microchemical Tests.* If the opportunity has occurred of examining the vagina, a quantity of mucous may be removed by means of a glass rod and smeared on glass slides for examination at once for living spermatozoa, and for other tests. If the stain be dry, the following method may be adopted:—A piece of the fabric with the stain is moistened with very dilute hydrochloric acid—one drop in 1½ fluid oz. of water—on a microscopic slide and the soaking is continued for from half an hour to five hours, depending on the age of the stain. The fabric is then removed with forceps and dabbed several times on to slides, avoiding tearing and much pressure. The liquid on the slides is allowed to dry in air, and the slide fixed by heat in the ordinary way. It is then stained by eosin and methylene blue or other counter-stain such as methyl green, in which case the hinder part of the head acquires a deep blue or green tint, whilst the front and

middle of the head and the tail are stained deep red. Sterile 0·9 per cent. NaCl solution mixed with about 10 per cent. by volume of glycerine may be used as a solvent instead of acidified water.

In many cases where the stain is well defined a scraping taken from the surface by means of a scalpel may show the presence of spermatozoa more readily than by the soaking method.

In order to render the spermatozoa more distinct under the microscope, the employment of a solution of iodine in water has been recommended. Iodine does not alter the size or shape, but causes the bodies to appear in stronger relief. The proportions of the ingredients which are recommended are iodine one part, iodide of potassium four parts, water one hundred parts. Iodine thus used gives a strongly marked yellow colour to animal and vegetable substances, while it does not alter mineral matters. It brings out the form of the spermatozoa in colour.

Another method of rendering these transparent bodies more visible has been adopted. It has been discovered on re-examining a slide on which the watery solution of a spermatic stain had been allowed to dry spontaneously, that many of these bodies which were only indistinctly seen while moist were now very prominent and distinct in their form, and those which before appeared tailless now assumed their complete shape and length. They became in fact more opaque and distinct by drying. The drying should take place slowly, *i.e.*, by covering the liquid on the slide with the microscopic glass and keeping it in a cool place.

Florence's Test. While certain of the slides are being stained for microscopic examination it is advisable to carry out microchemical tests on one or more of the preparations. Florence, in 1896,[1] described a test in which the addition of iodine to seminal fluid produced a crop of crystals.

The test is carried out as follows :—

A drop of the soak from the alleged seminal stain is placed on a glass slide and allowed to dry or nearly dry. A cover slip is placed over this and a drop of Florence's solution (iodide of potassium 1·5 grammes, iodine 2·5 grammes and water 30 c.c.) allowed to run under the slip. The preparation is at once placed under the microscope, and if semen is present a crop of crystals similar in colour and shape to hæmin-crystals make their appearance round the edge of the preparation and gradually extend. At first they are small, but in a few minutes many of them reach an enormous size. They are disposed singly and in clusters, rosettes and all varieties of groups. They are not permanent and soon become unrecognisable.

It was at first thought that the test was specific, but it is now known that the crystals are produced by the action of iodine on choline, a natural base occurring in many cells. The test is, however, of great value, for it is always given in a positive case, and in a very large experience in examining all kinds of stains in specific cases and in experimental work I have never been able to obtain a positive Florence test except from seminal matters.

[1] "Arch. d'Anthrop. Crim.," 1896.

Berberio's Test.[1] This is a similar test to the above; the test fluid is a saturated watery solution of picric acid, and when added to a seminal stain a crop of crystals like Charcot-Leyden crystals are obtained.

4. *Microscopical Examination of Spermatozoa.* Living spermatozoa move for many hours out of the body when kept at a temperature of 98 F., and even retain their rapid motions when the spermatic liquid is mixed with water; but these motions cease immediately on the addition of urine or chemical reagents. The spermatozoa may retain vitality (or free motion) in the body of a woman for a short period, and movement should always be looked for in wet specimens. The actual time that spermatozoa may survive after ejaculation cannot be precisely defined, but if movement can be observed the ejaculation is certainly very recent.

The spermatozoa are best seen in a good light, with a power of 500 diameters; the head is ovoid and flattened—sometimes rather pointed; the tail is from nine to twelve times the length of the head. Micrometrical measurements of human spermatozoa show them to be 50–55 μ in length, the head being 4·3–5·2 μ long and 2·9–3·6 μ wide in its greatest diameter. A normal ejaculation contains about 500 millions of spermatozoa sperm cells in various stages of development, imperfectly formed spermatozoa, granular bodies and epithelial cells. Fibres of cotton, linen, or wool, and other substances, may be also mixed with them; and they may be associated with pus, mucus, and blood-corpuscles, and it is essential in examining a stain to note carefully the different types of cells, organisms, etc., in the preparation. Their form is so peculiar that, when once well seen and examined, they cannot be mistaken for any other substance, vegetable or animal, nor, with ordinary care, can any vegetable fibres be mistaken for them.

Is this Stain Produced by Semen?

It may be at once stated that the preliminary naked eye examination and the application of heat for the odour are absolutely valueless standing alone, and the only way in which a *positive* answer can be given is by the discovery under the microscope of *at least one unbroken* spermatozoon; if unmistakable in appearance, one such is sufficient.

The microscopical detection of spermatozoa in dry stains is attended with some difficulty when the stained stuff has been much rubbed or worn, or is of very coarse nature. The heads are too much like cellular debris, and the tails too much like minute fibres, to allow an opinion to be based upon fragments only.

The detection of dead or motionless spermatozoa in stains may be made at long periods after emission, when the fluid has been allowed to dry. Koblanck, made experiments on this subject, in reference to different periods of time, and found these bodies distinct after twelve months. The number of distinct and perfect bodies diminished according to the length of the period at which the examination was made. Thus, at the end of a year, only two perfect specimens could be perceived. Stevenson found them after a period of five years.

The length of time during which spermatozoa may be detected depends on the conditions to which the stains have been exposed. Stains on clean clothing which have been allowed to dry quickly and which have not

[1] *Med.-Leg. Jour. of New York*, 1905-6.

been roughly handled may last for many years, but stains which have recurred on dirty clothing in which some decomposition has occurred, or on clothes which have been roughly handled are soon unidentifiable.

Does the Absence of Spermatozoa negative the Stain being seminal ?

An opinion to the effect that " No spermatozoa—not semen," has been expressed by a medical jurist of some repute. It is not necessary to refer to the extraordinary difficulty of proving a negative in experimental work, for there are well-recognised conditions under which spermatozoa are absent from seminal fluid ; thus, they may not be found in the very young, the very old, or in those who are labouring under long-standing disease of the testicles. Even in the cases of healthy married men, who have had children, spermatozoa are not always found in the spermatic secretion ; their presence and number are subject to great uncertainty. Exhaustion from frequent intercourse, or constitutional causes, without actual bodily disease, appears to influence their production. There are also various other conditions in which they are not found ; these have been fully described by Hotchkiss[1] and Siegler.[2] The discovery of spermatozoa in stains on articles of clothing demonstrates that the stains have been produced by spermatic liquid ; but it must be most emphatically stated that the non-discovery of spermatozoa in a given stain does not prove that the stain is not seminal.

Biological Tests for Semen

The precipitin test may be applied to the stain extract in the same way as in testing for the origin of blood. The group of the semen may also be ascertained by tests similar to those employed on blood-stains, but it must be remembered that group-specific substances will be present only in the semen of secreting individuals of Groups A, B and AB. An absence of both group-specific substances from a seminal stain does not permit of a deduction as to whether the stain is from a group O person or from a non-secretor. Spermato-precipitins may also be prepared for the identification of semen. Details of the technique to be used are described by Smith and Glaister. (*see* Recent Advances in Forensic Medicine, J. & A. Churchill, Ltd. 1939).

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[1] Hotchkiss, *Fertility of Man*, and
[2] Siegler, *Fertility in Woman*, Wm. Heinemann, London, 1945.

CHAPTER VIII

ABORTION

To a lawyer this word has a somewhat different meaning from that which it has to a medical man : to the latter it is merely an untimely emptying of a uterus which contains the products of a conception ; to the former the word conveys the idea of a criminal offence, although such may not have been committed, for it is only the artificial emptying (or attempts to empty) the uterus which is a crime, and many abortions are produced by mere disease and accident.

In a medico-legal work, however, the subject must be thoroughly discussed from every point of view, and it is treated as follows :—

The Law as to Abortion.
The Natural Causes of Abortion.
The Artificial Production of Abortion which may be justifiable or criminal.
The Evidence to be obtained of Criminal Abortion.
Feigned Abortion.
The Abortion of Moles and Monsters, Extra-uterine Foetation.

The Law as to Abortion

The law relating to attempts to empty a pregnant (or a non-pregnant) uterus is contained in the Offences against the Person Act, 1861, sects. 58 and 59 of which are as follows :—

Sect. 58. *Every woman, being with child, who, with intent to procure her own miscarriage, shall unlawfully administer to herself any poison or other noxious thing, or shall unlawfully use any instrument or other means whatsoever with the like intent, and whosoever, with intent to procure the miscarriage of any woman, whether she be or not with child, shall unlawfully administer to her or cause to be taken by her any poison or other noxious thing, or shall unlawfully use any instrument or other means whatsoever with the like intent, shall be guilty of felony, and being convicted thereof* ***shall be liable to be kept in penal servitude for life.***

Sect. 59. *Whosoever shall unlawfully supply or procure any poison or other noxious thing, or any instrument or thing whatsoever, knowing that the same is intended to be unlawfully used or employed with intent to procure the miscarriage of any woman, whether she be or be not with child, shall be guilty of a misdemeanour, and being convicted thereof shall be liable to be* ***kept in penal servitude.***

The statute uses the word "miscarriage" only, and includes in that term, comprehensively, the emptying of a pregnant uterus at any time, and ignores altogether the terms "abortion," and "premature confinement," which are convenient descriptive words commonly used in medicine.

Throughout both sections the word "unlawfully" is used without any attempt to define its meaning, and as the statute nowhere defines a "lawful" emptying of the uterus, it is important for medical men to remember this when they contemplate emptying a pregnant uterus for, what seems to them, good and sufficient reason.

In attempts by a second person to empty a uterus the statute makes no difference in the nature of the crime if the uterus does not contain the products of impregnation. It would seem that the woman herself commits no crime by an attempt to empty her uterus when it does not contain a fœtus, although if it contains one, she commits a crime.

In *R.* v. *Lumley*[1] it was held that if, when the abortionist did the act, he must, as a reasonable man, have contemplated that death or grievous bodily harm was likely to result, he would be guilty of murder; but that if he could not, as a reasonable man, have contemplated either of those consequences, he would be guilty only of manslaughter.

A person charged under section 58 of the above Act may be convicted of the felony of child destruction under the Infant Life (Preservation) Act, 1929. (See below.)

CHILD DESTRUCTION

"The killing of one not 'in being,' *i.e.*, a mere unborn child, was until 1929 punishable neither as murder nor as infanticide when committed by a mother. There can be no murder of a child which dies before being born or even whilst being born, only of one that has been born, and, moreover, been born alive. For purposes of criminal law mere *birth* consists in extrusion from the mother's body, *i.e.*, in having 'come into the world.' If but a foot be unextricated, there can be no murder; the extrusion must be complete; the whole body of the infant must have been brought into the world. But it is not necessary that the umbilical cord should have been severed. And to be born alive the child must have been still in a living state after having wholly quitted the body of the mother. Hence that life still existed must be actually proved, and this may be done by giving evidence of any cry, or breathing, or pulsation, or movement, after extrusion. But it is not necessary that the child should have continued to live until it was severed from the mother; or even until it could breathe; for a child may perhaps not breathe until some time after full extrusion (though, on the other hand, infants sometimes breathe, and even cry, before they are fully extricated). A Parliamentary Committee of 1893 recorded the sapient verdict of a coroner's jury upon 'a child found dead, *aged about three months*; but no evidence as to whether or not it had been born alive.' Thus to constitute murder, the birth must precede the death; but it need not also precede the injury. Thus an act which causes a child to be born

[1] 22 Cox, C.C. 635.

much earlier than in the natural course, so that the child is rendered much less capable of living when born and accordingly soon dies, may itself amount to murder." [1]

A great encouragement to infanticide was afforded by the rule that *partial* extrusion was not sufficient. There was therefore passed the Infant Life Preservation Act, 1929, by which any person who with intent to destroy the life of a child capable of being born alive, by any wilful act causes a child to die before it has an existence independent of its mother, shall be guilty of the felony of child destruction, and shall be liable to penal servitude for life.

The Act contains the important provision that no person shall be found guilty of this offence unless it is proved that the act which caused the death of the child was not done in good faith for the purpose only of preserving the life of the mother. The value of this provision will be apparent to every medical practitioner. It is also enacted that evidence that a woman had at any material time been pregnant for a period of twenty-eight weeks or more shall be *primâ facie* proof that she was at that time pregnant of a child capable of being born alive. [cf. *R.* v. *Bourne* (*infra*)].

Professional Secrecy in cases of Abortion

The sixth edition of this work contained a report of the Royal College of Physicians as to the duties of medical men in cases of abortion. In July, 1913, difficulties had arisen, particularly at the Hampstead Infirmary, on the question whether the medical officers ought to report the occurrence of abortions in the infirmary which were the result of illegal operations performed on the patients before admission, or whether to report such cases would be a violation of professional secrecy. The Royal College of Physicians was asked to make a pronouncement on the professional question involved. Then at the Birmingham Autumn Assizes, December, 1914, the following case occurred which raised the whole question in an acute form.

A woman, upon finding herself pregnant, went to an abortionist, miscarried on September 19th, 1914, developed septicæmia in a week's time, was removed to hospital, and died suddenly on October 16th. Her relations knew that she had had an illegal operation. To one of the medical men who attended her she communicated, under secrecy, the name of the abortionist. The coroner's court returned a verdict of wilful murder. The magistrates, however, held that the evidence was insufficient for committal. The judge, when the case came before the grand jury, upheld this decision. Further, he considered that the lack of evidence was due to the fact that the doctors had not reported the case to the police, and on this point made the following observations to the grand jury :—

CHARGE OF MR. JUSTICE AVORY to the Grand Jury on the Case of *R.* v. X.

The learned judge said :

" This woman has been committed on the coroner's inquisition on a charge of murder. The magistrates who have investigated the case have dismissed the charge. The deceased woman, according to the evidence, clearly died as the result of an illegal operation. Three medical men in succession attended her, and to one at least she confided the name of the person who performed the act. No information was given to the police or the authorities, and the woman died without the deposition being taken or without any statement being made by her on her death-bed which could be used in a court of law. With the exception of a letter,

[1] Kenny's " Outlines of Criminal Law," 15th Edit., pp. 143–4.

which was found amongst deceased's papers, apparently making an appointment with the accused woman, there is absolutely no evidence against this accused person of having performed any operation upon the deceased woman, and the magistrates, taking that view, dismissed the charge. I need not remind you that any statement made by the deceased woman to a medical man is not evidence in a court of law. I can see no evidence, as the case now stands, which will justify you in finding a true bill against the prisoner for murder. The law provides that in the case of any person who is seriously ill, and who, in the opinion of a medical man, is not likely to recover, the evidence of such a person may be taken by a justice of the peace. Under circumstances like those in the present case, I cannot doubt that it is the duty of the medical man to communicate with the police, or with the authorities, in order that one or other of those steps may be taken for the purpose of assisting in the administration of justice. No one would wish to see disturbed the confidential relation which exists, and which must exist, between the medical man and his patient in order that the medical man may properly discharge his duty towards his patient: but there are cases, of which it appears to me that this was one, where the desire to preserve that confidence must be subordinated to the duty which is cast upon every good citizen to assist in the investigation of a serious crime, such as is here imputed to this woman. In consequence of no information having been given, it appears to me, that there is no evidence whatever upon which this woman can properly be put upon her trial.

"I have been moved to make these observations because it has been brought to my notice that an opinion, to which I was a party some twenty years ago, when I was at the Bar, has been either misunderstood or misrepresented in a text-book of medical ethics, and I am anxious to remove any such misunderstanding, if it exists.

"It may be the moral duty of the medical man, even in cases where the patient is not dying, or not unlikely to recover, to communicate with the authorities when he sees good reason to believe that a criminal offence has been committed. However that may be, I cannot doubt that in such a case as the present, where the woman was, in the opinion of the medical man, likely to die, and therefore, her evidence was likely to be lost, it was his duty, and that some one of those gentlemen ought to have done it in this case."

Thereupon the Royal College of Physicians took a great deal of trouble to obtain information, discussed the matter fully, took counsel's opinion on some legal points, and finally passed the following resolutions for publication to those concerned, *viz.*, the medical profession:—

ROYAL COLLEGE OF PHYSICIANS OF LONDON

Resolutions concerning the Duties of Medical Practitioners in Relation to Cases of Criminal Abortion

The College is of opinion—

1. That a moral obligation rests upon every medical practitioner to respect the confidence of his patient; and that without her consent he is not justified in disclosing information obtained in the course of his professional attendance on her.

2. That every medical practitioner who is convinced that criminal abortion has been practised on his patient, should urge her, especially when she is likely to die, to make a statement which may be taken as evidence against the person who has performed the operation, provided always that her chances of recovery are not thereby prejudiced.

3. That in the event of her refusal to make such a statement, he is under no legal obligation (so the College is advised) to take further action, but he should continue to attend the patient to the best of his ability.

4. That before taking any action which may lead to legal proceedings, a medical practitioner will be wise to obtain the best medical and legal advice available, both to ensure that the patient's statement may have value as legal evidence, and to safeguard his own interest, since in the present state of the law there is no certainty that he will be protected against subsequent litigation.

5. That if the patient should die, he should refuse to give a certificate of the cause of death, and should communicate with the coroner.

The College has been advised to the following effect :—

1. That the medical practitioner is under no legal obligation either to urge the patient to make a statement, or, if she refuses to do so, to take any further action.

2. That when a patient who is dangerously ill consents to give evidence, her statement may be taken in one of the following ways :—

(*a*) A magistrate may visit her to receive her deposition on oath or affirmation. Even if criminal proceedings have not already been instituted, her deposition will be admissible in evidence in the event of her death, provided that reasonable written notice of the intention to take her statement was served on the accused person and he or his legal adviser had full opportunity of cross-examining.

(*b*) If the patient has an unqualified belief that she will shortly die, and only in these circumstances, her dying declaration will be admissible. Such a declaration may be made to the medical practitioner, or to any other person. It need not be in writing, and if reduced into writing it need not be signed by the patient nor witnessed by any other person, though it is desirable that both should be done, or that, if the patient is unable to sign, she should make her mark. If possible, the declaration should be in the actual words of the patient, and if questions are put, the questions and answers should both be given, but this is not essential. If the declaration cannot there and then be reduced into writing, it is desirable that the person to whom it is made should make a complete note of it as soon as possible.

January 27th, 1916.

It must not be supposed that the above is now the fixed law of England, but the resolutions may be taken as representing an attitude which if adopted by a medical man would certainly absolve him from any censure by the College and would probably gain for him any support which the College or any medical protection society could offer.

Both the *Lancet* and the *British Medical Journal* discussed the matter in vol. 1, 1916, *q.v.* The statements of Avory, J., appear to have been *obiter dicta* and merely set out his opinion for what it was worth.

Illness and Disease as Causes of Abortion (More Medico)

If a medical witness be asked the question, " Was this uterus emptied as the result of disease or illness ? " it is impossible by physical examination alone to answer specifically ; he is obliged to depend upon circumstantial or direct evidence as to the nature, or the existence, of disease. In quite recent delivery he may for instance find the woman still suffering from an acute fever, but in the majority of cases he will have to depend upon his ordinary professional knowledge as to the tendency of certain diseases, if present, to cause abortion.

Simons[1] in an analysis of 1,000 abortions showed that abortions both spontaneous and induced were more common in the first eight weeks of pregnancy. They were distributed as follows :—

	1,000 *Abortions.*			
Month	Spontaneous	Self-Induced.	Criminal.	% total.
1	64	25	3	9·2
2	249	158	18	42·8
3	203	119	17	34
4	33	34	8	7·5
5	35	18	2	5·4
6	7	2	0	0·9

[1] Simons, J. H., *Amer. Jour. Obs. and Gynae.*, 35, 571 : 1938.

According to Whitehead[1] one pregnancy in 4·7 terminates in abortion from natural causes.

It is impossible in a medico-legal work to give a complete list of all these, but the following are well recognised :—

Specific Fevers of all kinds. During an attack of fever, the uterus may empty itself; in quite recent cases definite evidence of the disease may be forthcoming.

Syphilis—an extremely frequent cause of abortion, especially in the second half of pregnancy, and without marked syphilitic symptoms other than the repeated abortions.

In *R.* v. *Wark*, the dead woman had suffered from syphilis three years previously, and this was urged by the defence as a possible cause of the abortion. Circumstantial evidence was given as to the use of a catheter, and, the deceased made a "dying declaration" that she had herself used it on her own person. A medical witness testified that death was due to septicæmia, and that there was nothing locally to show whether the abortion was due to disease or to instrumental interference, or even to the *lawful* action of another medical man in an attempt to save the life of a woman who was dying of septicæmia. He did not know of any case where a woman had used a catheter on herself (*vide infra*). The accused was found guilty, sentenced to death, but this was subsequently reduced to penal servitude.

Toxæmias of Pregnancy, eclampsia.

Excessive Vomiting of Pregnancy.

Severe Anæmias, Diabetes, Hypothyroidism, and other Endocrine Dysfunction.

Nephritis.

Advanced Heart, Lung or Liver Disease may cause abortion, and are certainly dangerous complications of pregnancy.

Uterine and Ovarian Abnormalities and Disease—these may arise from an abortion or badly managed delivery, but once established they may be the cause of further abortions.

Disease of the Placenta—syphilitic disease, extensive infarction, endarteritis other than syphilitic, accidental separation, hæmorrhage.

Abnormalities of the Decidua.

Disease, Maldevelopment and Death of the Foetus

These may be associated with any of the above material causes; if once established, they almost inevitably lead to abortion.

Deficient Diets.—the prolonged use by the mother of a diet deficient in lime, iodine, vitamins, etc., may so affect the vitality of the germ cells that proper development may not take place, or early abortion may occur.

Defective spermatozoa, defective ova, incompatibility of ovum and sperm, and incompatability of the blood of husband and wife, have all been suggested as possible causes of abortion.[2]

[1] Proceedings Royal Society of Medicine, Section of Obstet., Dec. 1929.
[2] Beck, *Obstetrical Practice*. Williams and Wilkins, Baltimore, 1942.

Justifiable (Therapeutic) Abortion

The law of England does not specifically recognise any interference with pregnancy as justifiable, even though the statutory references to the *unlawful* use of instruments, etc., might be held to imply that there was such a thing as lawful use. A medical practitioner must remember this when he contemplates emptying a pregnant uterus. The only reasons which provide a satisfactory justification for the induction of labour are two in number, namely, to save the life of the mother and to save the life of the child. As the law stands at the present time, interference with pregnancy cannot be justified on economic or ethical grounds.

There was a discussion on the indications and methods for the induction of abortion by the Section of Obstetrics and Gynæcology at the Congress of the British Medical Association in 1926.[1] The majority of the Section appeared to agree that abortion should be advised only for some definite morbid condition, such as tuberculosis, chronic nephritis, heart disease, pyelitis, toxæmic vomiting, vesicular mole, etc., which endangered the life of the mother. No one of these conditions could be regarded as a reason for always terminating pregnancy, but in the particular case any one of the said conditions might make it necessary.

As to the methods advocated, it appeared to be the general opinion that up to the third month or slightly less the cervix should be dilated and the ovum removed at once, or hysterotomy performed. After this period the slow method was advocated—that is, by rupturing the membranes ; by plugging the vagina and cervix ; by dilatation of the cervix by laminaria tents, metal dilators, or by the small hydrostatic bag ; by introduction of soft rubber bougies into the uterus or the introduction of a stomach tube.

To prevent a false charge, a chance of blackmail, and even to prevent misapprehension on the part of a woman, a medical man should, before emptying a uterus, place himself in the position of being able to prove that he did it for one of the two reasons given above, *and for no other*. Details of the actual grounds for belief that life can be saved by the act would here be out of place ; they can be found in any good work on obstetrics ; suffice it to say that pelvic deformities, chronic heart and lung disease, and vomiting of pregnancy constitute the bulk of them.

The golden rule is **never to empty a uterus without first having a second professional opinion as to its necessity ;** if this opinion be adverse, do not do it ; if it be favourable, it is well to get it in writing, and it is well also to get the written or attested consent of the woman and her husband or guardian, and then proceed to do it with all the skill and care possible. The death of the fœtus is at any time the most certain means of causing the womb to empty itself, but after the sixth month the operation is performed usually with a view to preserving this life, and steps must be taken accordingly.

Parturition may sometimes take place safely at the full time, even when the deformity of the pelvis is apparently so great as to lead many accoucheurs to conclude that natural delivery is impossible. Hence a cautious selection should be made, because the operation is necessarily attended with some risk ; and it does not ensure safety to a woman

[1] *B.M.J.* Aug. 7, 1926.

and the child. It should place the woman in a better position than she would be in if the case were left to itself; and of this the practitioner should feel assured. In the event of the death of the woman or the child, he exposes himself to the possibility of a prosecution. If the child were born alive, and died merely as a result of its immaturity, this might give rise to a charge of manslaughter.

Surgeons are sometimes charged with unlawfully procuring abortion under the Offences against the Person Act, 1861, sec. 58. In *R.* v. *Bourne*,[1] a surgeon of the highest skill openly in a London hospital performed the operation of abortion upon a young girl not quite fifteen years of age who was pregnant as a result of rape. The judge said that the surgeon was not obliged to wait until the patient was in peril of immediate death, but that it was his duty to perform the operation if on reasonable grounds and with adequate knowledge he was of opinion that the probable consequence of the continuation of the pregnancy would be to make the patient a physical and mental wreck. The surgeon (who was acquitted) said that no surgeon would venture to act except after consulting some other member of the profession of high standing, so as to confirm his view that the circumstances were such that an operation ought to be performed, and that the act was lawful.

CRIMINAL ABORTION

Abortionists possess varying degrees of skill, from the black sheep of the medical profession (who may perform the deed *secundum artem*), through the midwife (who has some acquaintance with the anatomy of the parts), down to the totally ignorant layman (who is interested in a particular case, but makes no practice of the act).

It is usually quite impossible by strictly medical evidence to detect the activities of abortionists. It can be done only by inquiry into motives, and fees, and surrounding circumstances. The means commonly employed are either—

A. Violence, which may be	(*a*) general	intentional	}	or a combination of the two—
		accidental		
	(*b*) local	skilled		
		unskilled		
B. Drugs			}	Violence and Drugs.

A. Violence—(a) General

(i.) *Intentional.* Among the mechanical causes may be mentioned severe exercise at golf, tennis, horseback riding, cycling, surf bathing, and similar methods of causing violent agitation of the body. No marks of violence would be apparent in such cases. Any physical shock sustained by the body may operate indirectly on the uterus. Blows or violent pressure on the abdomen are sometimes resorted to, and in these cases the marks of violence may be perceptible.

(ii.) *Accidental.* This can only be separated from intentional by general evidence. Playing violent games in an advanced state of pregnancy might produce abortion.

[1] [1939] 1 K.B. 687.

Tidy[1] mentions also copious bleedings (in the seventeenth century), and over-tight lacing, as having been resorted to. He remarks that such means usually fail in their purpose, and not only so, but leave their traces in view for all to see, and quotes the two following cases in illustration :—

(1) In the Assize Court of the Loire-Inférieure, it was proved that a peasant, who had seduced his servant, and wished to make her abort, mounted on a strong horse and put the girl on the same horse, then galloped wildly hither and thither, throwing her down on the ground whilst in full gallop, and this repeatedly. Having tried this twice without success, he conceived the horrible idea of applying to her stomach bread just taken from a very hot oven. This means, like the former, failed, the poor victim ultimately giving birth to a living and well-formed child at term.[2]

(2) A case quoted by Dr. Guibaut of a young Munich lady, living in California. Becoming pregnant, she wished to go to Munich to be delivered. In crossing the Isthmus of Panama a railway collision occurred. In consequence of this, labour pains set in. In spite of this she embarked for Portsmouth. She had a horrible passage, with further accidents. Notwithstanding these the pains subsided each time. On reaching Paris, she fell from the top to the bottom of the hotel stairs ! Again she was seized with pains like those of labour. She was then eight months pregnant. Next day she departed for Munich, and was not confined till some days after her arrival in that city.

(b) Local

(a) Skilled—leaving no traces of violence. Modern life has brought with it an amount of nervous and physical degeneracy of such a nature that an increasing number of women are unable to carry children to full term. Medical science has consequently been busy in perfecting the means of safely emptying a uterus when the life of the mother or the infant demands it. The use of dilators, Barnes' bag, metal sounds, and the introduction of soft rubber bougies, etc., combined with the very strictest attention to asepsis, have rendered the operation less risky, and usually free from signs that a medical jurist could detect as evidence of malpraxis. The danger in the criminal use of such means lies in haste. Sudden death from inhibition sometimes occurs when a sound is introduced into the uterus or when a jet of fluid or air is forced into it by means of a syringe.

When the membranes are penetrated and the waters are discharged, uterine action is invariably induced, but the time which elapses from the performance of the operation to the commencement of labour is subject to great variation. Ramsbotham states that he has known the uterus begin to act in *ten hours* after the rupture, but in another case a week elapsed before its action commenced. As a general rule, uterine action is fully established in fifty or sixty hours. It must not be supposed, however, that where a criminal intention exists so long a period is required for removing the contents of the uterus. In a criminal attempt by a medical practitioner, in which the woman would be a consenting party to the act, the removal of the embryo or fœtus may be, and generally is, effected in a much shorter period of time. At any rate, the time for the completion of abortion could not be measured by cases in which the uterus had been left to undergo spontaneous contraction after the membranes had been punctured and the waters had escaped. There might, however, be great danger to a woman in the necessary manipulations.

[1] *Leg. Med.*, pp. 164, 165.
[2] Brillaud-Lacjardiere, "De l'Avortement Provoqué," Paris, 1862, p. 279.

Cases in which medical men have thus lent their aid are unfortunately only too common. In his address to the jury at the Central Criminal Court, the Recorder of London referring to the case of a medical man who was charged with the illegal use of instruments, said

"The circumstances of this last case disclose a condition of morality which, I trust, has not spread very far or very wide. If it does there must ensue a decadence of our race, which must have most serious consequences. Had the woman in this case died, the doctor would have been charged with murder. Of late there has been a tendency in such an event to minimise this offence to manslaughter, but by what process of reasoning this course is arrived at I have not been able to understand."

Midwives from their calling acquire considerable skill in procuring abortion, and it is to be feared that they lend themselves in some instances only too readily to the practice.

Some doubt was once expressed in regard to the precise way in which skilled instrumental interference brings about abortion.

In *R.* v. *Bower* a sound was said to have been passed by the prisoner, and the ovum was said to have been expelled entire, *i.e.*, still enclosed in its membranes, with placenta attached. For the defence it was alleged that abortion could not have been procured by simply passing a sound without **rupturing the membranes.** This defence rested upon a statement to the required effect appearing in a work on Medical Jurisprudence. Such a defence is absolutely without basis in medical fact.

It is possible that a bougie, catheter, or similar instrument, may be passed into a pregnant uterus without abortion following, but such an event would be unusual: this might happen in the very early days because the instrument had not disturbed the ovum in any way. It might happen in the later stages because the womb was particularly healthy, and soon repaired the injury done. Abortion does, as a rule, occur, because either the membranes happen to be ruptured, leading to death of the fœtus; or a portion of the placenta is separated, and the uterus is too weak or irritable to repair the damage; or finally, it may happen because the uterus is stimulated to contract by the introduction of anything through its cervix. These statements are based on the universal consensus of opinion of all reputable obstetricians, and they are directly at variance with the above-mentioned defence.

If the membranes are ruptured abortion is practically inevitable; but this does not invalidate the fact that rupture is not indispensable for abortion to occur.

(b) Unskilled—leaving traces. It is sometimes assumed that it is impossible for a woman to induce abortion by instrumental means upon herself. Evidence is given below that such is not necessarily the case, but for all that it is the rule that at least a second person is concerned. When such second person is a medical man, the very fact of injuries being found argues want of skill, or secrecy and haste, and suggests an evil intent, though such must not be taken for granted, for when the operation is legitimately undertaken slight lacerations may occur. Such traces of violence must not therefore be taken to *prove* criminality till all the circumstances have been investigated, however much they may suggest it.

Even a medical man may sometimes cause injuries which cannot be accounted for on any innocent hypothesis.

In *R.* v. *Stadtmühler* a German physician was charged with murder. He was consulted by a healthy young woman, *æt.* 22, as to her pregnancy. He employed instruments for the purpose of procuring abortion. She died within forty-eight hours, and on inspection, severe internal injuries were found, which fully accounted for her death.

In a Scottish case, the uterus near its mouth presented two openings in its substance, described as punctured wounds by the medical witnesses for the prosecution who made the examination, and as the openings of torn blood vessels by others who were called for the defence. There was also a rupture of one ovary. The abortionist was convicted; but the medical man who was believed to have been the principal agent in the crime committed suicide.

Any apparent mechanical injury to the womb should be minutely examined at the time of inspection, so that no doubt of the cause may afterwards be entertained.

The professional abortionists of India, the native *dhaees*, who are women of the lowest castes, generally adopt the following method of procuring abortion. They insert into the uterus a twig of a tree about six or eight inches long, smeared with asafœtida. The membranes are ruptured, abortion takes place, and if the woman dies from peritonitis, the walls of the uterus will usually be found perforated. It is a common practice in these cases to refer death to snake-bite, in order to prevent inspection of the body, which generally reveals the means by which the abortion was procured.

A case was tried some years ago in which the evidence showed that the prisoner had attempted to produce abortion in the deceased by thrusting wooden skewers into the substance of the uterus. Inflammation and gangrene took place, and the woman died. The prisoner was convicted of murder and executed.

A case was once treated in the London Hospital in which the bladder had been perforated by an abortionist. The criminal was never detected.

In *R.* v. *Buckley* a man was charged with the murder of his wife, aged about 42. The medical man's account of the evidence is as follows:

I found the deceased in a friend's house lying upon a sofa. I was informed that she had run from her own house to the house where I found her (a distance of two hundred yards), to escape a renewal of her husband's violence. I made an examination of the deceased. She was dressed in her night clothes. There was slight hæmorrhage from the vulva; the inside of the vagina had blood trickling down, and contained a few dark-coloured clots: the patient complained of great pain in the abdomen, and was suffering from shock to the system. There were no signs of commencing labour, and the membranes were intact; the mouth of the womb was patulous. A midwife in attendance informed me that the patient "was expecting confinement in a week." The patient was in a fit state for careful removal. In the presence of prisoner, I said to the deceased, "Your husband is here; tell me, what has he done to you?" She replied, "He threw me on the bed, and put his hand in me, many a time." The prisoner replied, "It's all lies." The patient was dressed, and carefully transferred to a trap, and removed to her brother's house. During the remainder of that day I saw the deceased five times. The hæmorrhage had ceased, but patient was very weak, suffering from shock and collapse; there was no sign of labour. Dr. Tyndall, of West Houghton, and I made examination, and this was the first time at which suspicion of a rupture through the upper part of posterial wall of vagina was entertained; but we could not, owing to the tenderness of the parts and the weak condition of patient, make a more thorough examination. I administered hypodermic injections of ether and brandy, as a result of which the patient partially revived, and the magistrate and myself put questions to her, the answers to which I wrote down on paper, and the following is a copy:—"Has

kicked her on the privates, knocked her on the bed, made her get up, and kicked her; put his hand inside her three or four times, struck her face, says he was drunk. Had been drinking long; said he would knock her head off. Had often ill-treated her before. He condemned her of another man. He said he could do it as well as any man or woman, but for folk talking." To the question, "Did he want to bring on confinement?" she answered "Yes." "In fear of death she adhered to truth of statement. All she has said is quite true. He actually put his hand in her body five or six times."

Autopsy showed the following appearances:—

Externally. There were no external marks of violence about the genital organs, but the right upper eyelid was blackened by ecchymosis.

Internally. The outer surface of the uterus showed very distinct signs of acute inflammation. The intestines formed an arch over the uterus, and thus were supported. The uterus was opened by a longitudinal incision and contained a female fœtus of complete gestation, the placenta was situated on the anterior uterine wall. The head of fœtus was the presenting part. The interior of uterus showed the same signs of inflammation of its surface as did the other surface. The os uteri was slightly dilated, and its margins presented a sloughy and uneven condition. The upper part of the posterior vaginal wall at its junction with the cervix uteri was ruptured, the aperture being sufficiently large to admit a man's hand or fist, and communicating directly with the general abdominal cavity. The rupture, in our opinion, was caused by the insertion of a foreign body with appreciable force. The peritoneal cavity contained a quantity of serous fluid, there was evidence of inflammation of the bowels situated in the lower part of abdominal cavity. The other organs of the body were healthy.

The cause of death was metritis and peritonitis (caused by the rupture described) with hæmorrhage and shock to the system.

The following case shows well the extreme injury that may be inflicted in attempts to procure abortion; the defence was successful, but probably would not be so on another occasion:—

In *R.* v. *Westworth* the prisoner's wife had been married to him for eight years, had borne three children, and was sworn to have said that "she would rather kill herself than have another," but there was no evidence to prove that she believed herself to be pregnant at the time of her death. The prisoner stated that he arrived home about midnight and found his wife lying on the sofa in the parlour, as he thought, drunk, and bleeding from the vagina; he immediately fetched a doctor, and with his assistance carried her upstairs to the bedroom, where she died next morning. At the *post-mortem* examination of the body, the following pathological appearances were noted:—(1) Between forty and fifty small bruises on the legs and arms—other accounts say on front and inner side of the thighs, and a large one on the hip—that they could not have been produced by falls, nor by the victim herself; (2) four wounds in the vagina: of these one was about half an inch deep, just inside the vaginal orifice, near the urethra, a second of about the same depth was an inch higher up, also on the front wall of the vagina, a third at the left-hand top corner of the vagina, penetrating the left broad ligament, causing much extravasation of blood, but not penetrating the peritoneum; a fourth, much larger than the others in calibre, passed out of the vagina between the uterus and bladder into the peritoneal cavity; it then penetrated the tissues in front of the spine, ran alongside the aorta, entered the peritoneum again near the left kidney, twice perforated the mesentery of the top of the jejunum, and passed through the left kidney; a second plunge, apparently through the larger hole in the vagina, had sent the instrument up to the pyloric end of the stomach, which was bruised but not perforated. The deceased was found not to be pregnant, and the uterus was quite healthy. Three medical witnesses said that the bruises could have been caused only by some heavy instrument such as the round head of a poker, as, though most of them were small, they penetrated deeply into the muscles; they also said that it was physically impossible for such internal injuries to have been self inflicted. Three other medical witnesses on the other hand said that the bruises might have been caused when the deceased was carried upstairs, and that the internal injuries might have been self inflicted from behind whilst the

deceased was lying on the sofa. A small brass poker with blood on the handle was found in the room, and was alleged to be the weapon with which the above wounds were inflicted. The prisoner was acquitted on a charge of murder.

Local Injections. The injection of fluid into the uterus by means of a Higginson's syringe, is a common method of procuring abortion. The fluid used may be plain water, soapy water, or solutions of potent drugs such as corrosive sublimate. Many cases of sudden death from such practices have come under our notice, and many cases have been published by other observers.[1] [2] Death may be caused by the sudden distortion of the organs producing shock and inhibitions of the heart, or air embolism may occur from opening up the venous sinuses. Death occurs sometimes with startling suddenness after only two or three contractions of the syringe bulb.

In September, 1919, at the High Court of Justiciary, Edinburgh, a woman was charged with causing the death of a young girl by injecting water into the womb with a syringe. The os was found dilated and the membranes partially detached. Death was attributed to hæmorrhage and shock. The accused was convicted of culpable homicide. In 1928 a man named Palmer, who practised as a medical electrician, was charged with causing the death of a young woman in his rooms. The *post-mortem* examination indicated that a quantity of soapy water had been injected into the uterus and that death was attributed to shock caused by the injection. Palmer was sentenced to seven years' penal servitude. In a case tried in Glasgow in 1933 it was proved that a strong solution of pearl-ash had been injected into the uterus of a young woman. An abortion took place followed by the death of the woman. At the autopsy the uterus was found to be profoundly damaged with complete destruction of the lower segment. The mechanical effect of an innocent liquid may be more effectual in producing abortion than the use of an irritating liquid. In medical practice, tepid water has been employed as an injection for the purpose of inducing premature labour. Lazarewitch has published twelve cases in which the injection of water at 95° F. caused the uterus to contract and expel its contents.[3] The earliest period at which Lazarewitch employed water was in the thirtieth week of pregnancy. In most of the cases the women had reached the thirty-sixth week of pregnancy. This is much later than the usual period at which abortion is commonly attempted for criminal purposes. An innocent injection may be used to produce abortion. The words of the statute, however, "other means whatsoever," appear sufficiently comprehensive to include the use of a non-noxious liquid, and according to the decision in *R.* v. *Wallis*, it is not material to prove that the liquid employed is *per se* of a "noxious" nature.

In *R.* v. *Collins* there was some evidence to suggest that the abortionist had used injections of corrosive sublimate to effect his purpose. This substance might be chosen in false reliance upon its undoubted antiseptic powers.

Self-induced Instrumental Abortion. The following case serves to emphasise the possibility of self-induced instrumental abortion and

[1] Muller P. Annales de Med Legale, Oct., 1939 : 643.
[2] *Brit. Med. Journ.* 1 : 921 ; 1939.
[3] "Trans. of the Obst. Soc.," vol. 9, p. 161.

the care necessary in dealing with such cases in private to protect oneself against, at least, the suspicion of malpractice.

"A married woman with several children sent for me to visit her, and gave the following history: She knew herself pregnant, having gone several days over her time; since then (several weeks) she had been losing blood constantly. On examining her the cervix was found soft and patulous, with blood escaping from it, and the uterus irregularly contracted, apparently about the size of the pregnant uterus at four months. She had no fever, pain, nor tenderness.

"On further questioning her, she at first admitted taking 'herbs'; then that she had 'scraped out the womb with a hairpin,' showing me the pin, which was an ordinary woman's hairpin, the looped end of which she had used as a 'curette,' the operation being rendered easier by her suffering from slight prolapse, and pressing on the fundus through the abdominal wall. She informed me that she had done the same thing twelve months ago, with the result that in the course of time she was delivered of what seems to have been a fleshy mole. She informed me that she had told no one but myself of the cause; and, with the possibility of complications in view, I decided to send her to hospital. Through the courtesy of the house-surgeon I am able to state that after a course of ecbolic medicine she was delivered of a fœtus of about five months' gestation, which appeared to have been some time dead, and the patient progressed satisfactorily. I am induced to chronicle the above case because it has been frequently stated that self-induced instrumental abortion is impossible."

A girl confessed that she had pushed the handle of a small paint brush up the womb till she "felt something give way"; abortion had resulted.

A curious case was tried at Exeter many years ago in which a vaginal speculum figured prominently.

One of the accused, who was a surgeon, had on several occasions passed a round polished instrument into the body of the woman, once in a coppice and at another time in a field. The defence was that the surgeon had merely used a speculum to ascertain whether she was pregnant in order to know how to prescribe for her; and that it was absurd to suggest that he had ever intended to procure abortion, for this had not followed, and that it might have been easily induced by him at any period of pregnancy if he had wished it. The prisoners were acquitted. It is well known that a speculum is not required at all for determining the question of pregnancy. No medical man who wished to induce abortion would ever make use of such an instrument; though it is conceivable that in an unhealthy uterus such vaginal manipulation might possibly cause the event, it would be to the last degree improbable that a healthy uterus would empty itself upon such provocation. But for all that, when this instrument has been improperly or unnecessarily used on a pregnant woman, a charge of an attempted abortion by instruments may be easily raised against a medical practitioner.

B. Production of Abortion by Drugs[1]

The following generalisation, which is strictly warranted by facts, conveys a warning to would-be abortionists, whether professional and habitual, or lay and occasional—**There is no drug and no combination of drugs which will, when taken by the mouth, cause a healthy uterus to empty itself without endangering the life of the woman who takes it.**

The action of reputed emmenagogues and ecbolics has been the subject of little experimental work, and the conclusions as to the action of certain drugs rest almost entirely upon clinical evidence, often of very doubtful value. *Emmenagogues* may be defined as remedies used to produce or increase the menstrual flow. They may be divided into

[1] See "Quacks and Abortion," *Lancet*, 1898, vol. 2, and 1899, vol. 1.

direct and indirect: the former are supposed to act directly upon the uterus or the nervous system in close relation to it, while the latter act by promoting or restoring the health of the body as a whole.

Indirect emmenagogues include, therefore: *Tonics*, such as iron and arsenic; *Hæmatinics*, especially iron; and *Purgatives*, such as colocynth, gamboge, magnesium and sodium sulphate, and aloes, croton oil, elaterium, hiera picra (a mixture of aloes and canella bark), and pilacotia (a mixture of aloes and colocynth).

Amongst direct emmenagogues the following drugs have from time to time been included: aloes, cantharides, caulophyllin, borax, apiol, cimicifuga racemosa, potassium permanganate, manganese dioxide, myrrh, anemone pulsatilla, polygala, senega, sanguinarin, pennyroyal or mentha pulegium, senecio, yew leaves, grains of paradise, tansy, hellebore (white and black), squills, broom, male fern, laburnum; asarum arabicum.

Ecbolics may be defined as drugs which cause the death or expulsion of the fœtus. Commonly included amongst this class are ergot, quinine, hydrastis canadensis, ruta, juniperus sabina, pituitary extract, lead and certain other metals such as mercury.

The native Indian abortionists employ the following drugs: camphor, the juice of the jaeta, the mulberry, and seajeenaroot, as well as pan root—a species of pepper. These act chiefly as irritants, although they are supposed to have a specific effect on the uterus as ecbolics.

Oil of Absinthe as a Supposed Abortifacient.[1] An inquest was held on the wife of a farm labourer who died three-quarters of an hour after swallowing over 100m of oil of absinthe, which she had obtained for the purpose of terminating pregnancy. She had obtained the drug from a firm of qualified chemists in Liverpool. It appeared that the drug came by the evening post, that the deceased took it almost immediately, and that a few minutes later she was found lying speechless in the yard. The jury returned the verdict that death was caused through deceased taking an overdose of oil of absinthe to cause abortion, and recommended that such a dangerous drug should be scheduled as a poison.

Aloes apparently act by producing congestion of the large intestine and of the pelvic organs. It is said to have a direct effect upon the uterus, but there is no evidence of this.

Anemone pulsatilla. There is clinical evidence to show that this drug is useful in amenorrhœa. It has an irritant effect upon the kidneys and digestive tract.

Apiol. The neutral principle of petroselinum sativum or common parsley has achieved a reputation as an abortifacient. It is reputed to have caused abortion in sixteen cases out of twenty in which it was used.[2] It appears to have a toxic effect on the liver but in certain cases where death has occurred after its use the fatal effect was due to tricresyl phosphate used as an adulterant.

Asarum Europœum Asarabacca. The powdered leaves of this plant were formerly used in medicine. The leaves, as well as the root, are

[1] *B.M.J.*, 1902, 2.
[2] *Annales de Méd. Légale*, November, 1937: 993.

irritant and acrid, owing to the presence of an essential oil They have an aromatic and bitter taste. In doses of from half a drachm to a drachm these preparations excite vomiting, purging, and griping pains. Like other acrid or irritant substances, they may lead indirectly to abortion by their effects on the general system, but they have no specific action on the uterus. Maschka met with the following case, in which a decoction of the leaves, taken by a pregnant woman, was followed by death without causing abortion.

A woman who had reached the fourth month of her pregnancy was advised to take a decoction of asarum for the purpose of exciting abortion. Pains in the abdomen were followed by convulsions, which proved fatal on the second day. The coats of the stomach and duodenum were found softened and reddened. The stomach contained a pasty-looking substance, without any appearance of leaves, roots, or seeds. The kidneys were much diseased, and in the uterus there was a four months' fœtus. The contents of the stomach were examined chemically, but nothing was found to throw a light on the cause of death. The fact that she had taken a decoction of asarum was rendered probable by the evidence of witnesses ; but it had not produced the usual effects of vomiting and purging. Maschka ascribed death to a diseased condition of the kidneys, leading to uræmic poisoning of the blood. This had, in his opinion, caused *eclampsia gravidarum* and death.

Atropa Belladonna. A medical man was convicted at the Central Criminal Court of Sydney of administering *extract of belladonna* as a suppository, in order to excite abortion ; this substance has no specific influence on the uterus.

Cantharides has no special effect upon the uterus, but has, however, caused abortion in large doses, although one drachm has been taken by a pregnant patient with no effect. It might produce abortion in large doses by its action as an irritant poison.

Caulophyllin, a resinoid powder obtained from the root. This principle is said to have a direct influence upon the uterus or upon the motor nerves supplying the uterus. It has been used in America for the purpose of producing abortion with apparent success. The dose of caulophyllin is given in Martindale's Extra Pharmacopœia as from one to four grains, but the dose necessary to procure abortion is not definitely known.

Ergot. As to the action of ergot there can be no doubt that it is a *true ecbolic.* The action upon the muscular tissue is mainly due to ergotoxine, ergotamine, and ergometrine, together with tryamine and histamine, which though present in many preparations of ergot are not true principles of that drug. Ergot produces powerful uterine contractions, and although it acts best when uterine action has started, yet given in a fair dose at the time when the menstrual periods should occur it would be likely to initiate uterine action and so in a case of pregnancy would act as an abortifacient.

If the dose is small, the rhythmic contractions of the uterus are strengthened, and if the uterus is at rest, contractions are initiated. With larger doses the contractions become more powerful, and last a longer time.

In powder, infusion, or tincture, it has been for some time used by medical practitioners to excite the action of the uterus and aid parturition. It is also used for a similar purpose on animals in veterinary practice.

In a case which occurred at Brighton, a question arose respecting the fatal effects of ergot on a woman who had taken it for a long period, obviously with a view to procuring abortion. She died, however, without abortion having taken place; and the question at issue was, whether this drug had or had not caused her death. The dose taken was about a teaspoonful of the tincture of ergot three times a day, for a period of eleven weeks. On inspection, patches of inflammation were found on the mucous membrane of the stomach after death. No other cause for death was apparent, and one medical witness assigned it to the poisonous irritant action of the ergot, as, at the early stage of pregnancy which she had reached (the third month), this substance would not be likely to act as an abortive. Another medical witness asserted that death could never be primarily caused by ergot of rye. Tardieu reported the case of a woman, *æt.* 24, who aborted in the fourth month of pregnancy, as a result of the administration of ergot in powder; she died from peritonitis in about twenty-four hours. The ergot was found in fragments in the lower third of the bowels. In *R.* v. *Brown* a woman was tried for administering ergot to a married woman with the view of procuring abortion. There was little doubt that ergot was the substance administered; and though repeated doses were given, the drug failed of its effect. The case broke down, the only evidence being that of the patient herself, and the judge directed an acquittal on the ground that the woman to whom the drug was given by consenting to the operation made herself an accomplice; and that it is a maxim of English law that no person can be convicted of a criminal offence on the unsupported evidence of an accomplice.

For actions and symptoms, other than ecbolic, *vide* "Poisoning by Ergot."

Pituitary Extract. This substance has a specific effect on uterine muscle, causing definite and powerful contractions. It acts only when injected into the body, and has no effect when given by the mouth. It is not likely to be used as an abortifacient, therefore, except by medical men or midwives.

Quinine. The definite effect produced by the administration of quinine during labour is partly due to its general tonic action and partly to a direct action upon the uterus or uterine nerves. It definitely increases uterine contraction, but there is no undisputed evidence that it will produce abortion even when pushed. Quinine and castor oil combined appear to cause a more marked uterine stimulation than quinine does when used alone.

Hydrastris Canadensis. This causes contraction of the peripheral blood vessels, and acts upon the uterine muscular tissue. It has a direct effect upon the muscular fibres of the uterus due to the hydrastine or hydrastinine it contains. In one case, 17½ grammes of hydrastine in five days brought on labour at the seventh month. It is of very little use as an emmenagogue, but is a definite ecbolic, and therefore might produce abortion.

Juniperus sabina, Savin. Savin is reputed to have an abortifacient action, but it is exceedingly doubtful if it has any direct effect upon the uterus. It is employed as a popular abortive. In small doses it is useless, while in large doses it acts as an irritant poison. The woman may die undelivered or the fœtus may be expelled, and the mother subsequently die from the effects of the drug.

The fatal irritant action of savin will be evident from the following case. The deceased, a healthy woman, had reached about the seventh month of pregnancy. She was very well on the Friday, but was seized with vomiting on the Saturday,

and she stated that she had taken nothing to produce this. The vomiting continued throughout Sunday and was of a green colour. She was first seen by a medical man on Sunday evening. The symptoms were those of inflammation of the stomach and bowels, with great anxiety: pulse 150. The green colour of the vomited matter was at first supposed to be owing to bile. The vomiting appears to have continued at intervals, but it does not seem that there was any violent purging. Labour supervened on Wednesday. The child was born living, but soon died. The woman died on the Thursday, *i.e.*, five days after having taken the poison, for there was no proof that any savin could have been taken after Saturday. The brain and lungs were healthy, except that the air-tubes had a dark red colour; the heart was flabby; the blood was generally fluid. The lining-membrane of the gullet was reddened, and had on it ecchymosed patches. Half of the mucous membrane, from the cardiac orifice upwards, presented a dark red arborescent injection, with slight patches of ecchymosis; but there was no erosion or ulceration. In the stomach a large patch of redness, about three inches wide, extended from the greater curvature towards the pylorus. The vessels of the mucous membrane were considerably injected, forming infiltrated patches, especially about the lesser curvature, extending towards the cardiac end; but there was no ulceration or erosion. The stomach contained nearly eight ounces of a greenish fluid, of the appearance and consistency of green-pea soup. By examining a portion of the washed vegetable substance under a microscope, and by drying a portion, rubbing it, and observing the odour, clear evidence was obtained that the green colour was owing to the diffusion of finely triturated savin powder (*vide* p. 713). The interior of the duodenum, especially towards the pylorus, was intensely inflamed, being of the colour of cinnabar. Patches of inflammation were found throughout the other portions of the intestines. There was some inflammation of the peritoneum, chiefly over the upper part of the bowel and omentum. The kidneys were inflamed, and of a dark red colour; the bladder was healthy. Green-coloured mucous matter, containing savin, was found in the duodenum, but not in the lower part of the intestines. The quantity of poison taken by the deceased could not be ascertained, but it must have been large. The quantity remaining in the stomach after five days, under frequent vomiting, was from twenty-five to thirty grains.

In another case a woman, eight hours after she had taken savin, was found lying on her back, insensible, and breathing stertorously. She had been suddenly seized with vomiting, and this continued for some time. Labour came on, and she died in about four hours, during a fit of pain. She appeared to be between the seventh and eighth months of pregnancy, and the child was born dead. On inspection, twenty-four hours after death, the brain was found gorged with black fluid blood. The stomach was paler than usual, excepting in one or two spots which were red, as if blood had been effused into the mucous tissue. It contained four ounces of an acid liquid of a brownish-green colour. This, on distillation, yielded an opaque liquid, from which a few drops of a yellow oil were separated by means of ether. Some sediment found in a bottle presented, under the microscope, the characters of powdered savin. There can be no doubt that this substance was the cause of death. The action of the poison appears to have been, in the first instance, like that of an irritant, and just before death like that of a narcotic.

The symptoms are not always those of an irritant. In some exceptional instances, as in the subjoined case, the action of the poison was spent on the nervous system:—

A young woman, advanced to the eighth month of pregnancy, secretly took this substance for an abortive. A medical man who was called to see her found her with the teeth tightly clenched, and unable to swallow. There were tetanic convulsions and the body was slightly arched forwards. First upon examining the contents of the stomach, as well as a bottle containing a mixture, part of which she had taken before death, a large quantity of savin (which was the cause of death), was found.

Under a fatal dose of this drug, sufficient to act as a poison, a woman even advanced so far as the eighth month of pregnancy may die without any effect being produced on the womb.

The powdered leaves are the form in which savin has been often given as a popular abortive, and the above cases show the dangerous effects to the woman and child. The leaves of savin are readily obtainable in gardens. They may be given in the form of infusion or decoction. The former is the most powerful. Savin may also be given as a tincture, or as an essential oil. In all these forms, in large or frequently repeated doses, it has an irritant action. The powdered leaves are not used in medical practice. The dose as an emmenagogue would be from five to fifteen grains—the medicinal dose of the oil is from two to six minims, and the tincture is from twenty minims to one fluid drachm. This holds the oil and resin dissolved. The leaves of savin may be identified by their peculiar odour when rubbed, and also by their appearance under the microscope.

Cases in which the oil of savin has been administered for the purpose of abortion are common.

In *R.* v. *Pascoe* a medical man was convicted of administering oil of savin to a woman with intent to procure miscarriage. He had given fourteen drops of the oil, divided into three doses, daily—a quantity, which, according to the medical evidence at the trial, was greater than should have been prescribed for any lawful purpose.

In *R.* v. *Moore* a man was tried for and convicted of administering oil of savin to a pregnant woman. It made her very ill, but did not produce abortion.

The *oil of savin* is obtained in the proportion of 2 or 3 per cent. by weight by the distillation of the tops. It has a yellowish colour, and the peculiar terebinthinate odour of the plant, by which alone it may be recognised. It may be separated from the contents of the stomach by agitating them with its volume of ether, in which the oil is very soluble. The ether may be afterwards removed by distillation. The odour of the oil is stated to have been perceived in the blood and in the cavities of the body. This may be regarded as the best test of its presence. The oil of savin forms a turbid mixture with alcohol. When treated with an equal volume of sulphuric acid it acquires a dark brown colour, and when this mixture is added to distilled water, a dense white precipitate separates.

Myristica Fragrans, *Nutmeg*. This common flavouring agent has some reputation amongst ignorant people as an abortifacient; needless to say, no such action is known to the profession. For cases and symptoms *vide* "Poisoning by Nutmeg."

Myrrh. This has been said to act directly upon the uterus, but if it has any action it is feeble and probably indirect rather than direct. It acts as a stomachic tonic. There is no experimental evidence as to its abortifacient or emmenagogic powers.

Pennyroyal, *Mentha pulegium*. This is a popular emmenagogue and abortifacient. It has neither emmenagogic nor ecbolic properties, and is not now employed for any purpose by medical practitioners.

Any notice of this substance here would have been quite unnecessary, but for the fact that in a trial for criminal abortion,[1] strongly abortive properties were incorrectly assigned to it; and it was described as a highly noxious substance.

[1] *R.* v. *Wallis*, 1871.

Polygala senega. This is used in the United States as an abortifacient. The dose is uncertain, and there is no evidence either clinical or experimental as to its action upon the uterus.

Ruta or Rue. An infusion of this drug or the oil is supposed to have abortifacient effects. The drug is often taken for this purpose, but there is no accurate knowledge of its alleged qualities. It is stated that repeated doses cause vomiting, epigastric pain and salivation, followed by delirium and collapse. Labour pains may start 12—20 hours after taking the drug. In Greece it is commonly used together with Savin.[1]

Tardieu reported three cases in which a strong decoction of rue produced abortion at the fourth, fifth, and about the sixth month of pregnancy respectively, and the women recovered.[2] Among the symptoms caused by rue when taken for the purposes of abortion are profuse salivation and great swelling of the tongue. Abortion has occasionally taken place in five or six days. Rue acts most powerfully when taken in the fresh state. The active principle appears to be a volatile oil, which gives the peculiar odour to the plant. The oil is most abundant in the seeds. In the event of the leaves being taken, the best evidence will be furnished by their botanical characteristics.

Saffron. A decoction of the dried stigmas of saffron (*Crocus sativus*) has been employed as a popular abortive but there is no evidence of any abortifacient effect. In modern medicine its chief use is to give colour and flavour to liquids. It has been observed that when administered to pregnant women, the yellow colouring-matter has been absorbed, and the fœtus *in utero* has been stained with it. This appearance in the body of the fœtus might lead to a suspicion of its use, although no injury to the woman may have resulted.

Sanguinarin. A resinoid powder obtained from the blood-root (sanguinaria canadensis). This is said to be an emmenagogue.

Senecio Ragwort. This drug is stated to have an action on the uterus but is not an ecbolic, at any rate in ordinary doses.

Sodii salicylas. This drug often causes hæmorrhages from mucous membranes and not infrequently brings on menstruation when given for rheumatism. This was definitely proved by a case which was under observation for two years. Apart from this tendency to produce hæmorrhage it has no action on the uterus and is not a true ecbolic.

Tanacetum vulgare. Tansy. Oil of tansy. This oil has acquired a reputation as an abortive agent, and has caused death in several instances. Pereira quotes a case in which half an ounce of the oil proved fatal. The symptoms were spasms, convulsive movements, and impeded respiration; no inflammation of the stomach or bowels was discovered upon dissection.

A teaspoonful of the volatile oil was taken by a girl in mistake for the essence. She complained of giddiness, and became insensible in ten minutes; convulsions came on, with frothing at the mouth, difficult respiration, and irregular pulse; and she died in one hour after taking the oil. A healthy-looking girl, *æt.* 21, took eleven drachms of oil of tansy about six hours after a hearty dinner. She was found insensible, and in convulsions, soon after she had taken the drug. She died in three hours and a half. A strong odour of tansy was observed in the

[1] *Annales de Méd. Légales*, Nov. 1937 : 993.
[2] "Ann. d'Hyg.," 1855, 1, 403.

breath before death, and on inspection in the peritoneal cavity, stomach, and even the interior of the heart. The uterus contained a well-formed fœtus about four months old, which did not, either in itself or its membranes, present any evidence of having been disturbed. In a third case, a woman but a few weeks pregnant took half an ounce of the oil, and did not entirely lose her consciousness until three-quarters of an hour had elapsed, although she was convulsed at intervals before that time. She died without abortion being produced, within two hours after taking the poison.

These facts show, that while oil of tansy possesses no specific action on the uterus, it is capable of acting as a powerful poison on the brain and nervous system. The oil may be recognised, either before or after distillation of the contents of the stomach, by its peculiar and penetrating odour. It is very soluble in ether, and this may be employed for its separation.

Metals and Mineral Substances as Abortifacients. Mineral poisons have been employed for the purpose of procuring abortion, often with a fatal result. Lead is the most common metal used for this purpose but other substances may be mentioned including phosphorus, arsenic, corrosive sublimate, bichromate of potassium, blue vitriol or sulphate of copper, copperas or ferrous sulphate, and preparations of ferric chloride. Metallic mercury has been used by the mouth strangely enough.

A man administered to a girl, *æt.* 20, in the third month of pregnancy, about four ounces and a half of metallic mercury. It had no effect on the uterus, but in a few days, owing to oxidation and absorption of the metal, tremors began to affect the right side of her body. Her gait became unsteady, and she stumbled in walking. These symptoms continued unabated for two months and then affected the left side. She lost the power of grasping things. She went the full time, and the symptoms had then almost disappeared.

These powerful poisons may produce acute or chronic symptoms and may destroy life without affecting the gravid uterus or its contents. A woman, *æt.* 22, had passed the fifth month of her pregnancy, and died, it was supposed, from the effects of arsenic. It appeared from the evidence that, with the view of producing abortion, she had been advised to take a large dose of arsenic. She suffered from severe vomiting and purging, and died in seven hours without having aborted. A large quantity of arsenic was found in the stomach.

Tincture of Ferric Chloride has frequently caused severe symptoms, and seriously injured health, without producing abortion.

In one case it was proved that this compound of iron had been given in large doses daily to a pregnant woman, for the purpose of exciting abortion. It had no such effect. The prisoner also gave to her cantharides in pills. The defence was, that these were proper medicines for the treatment of amenorrhœa, under which it was alleged she was labouring. The large doses administered, and the secrecy with which the medicine was supplied, proved that they had been given unlawfully and with criminal intent; the druggist who supplied them was convicted.

Lead. This substance appears to have a specific toxic action on the cells of the developing ovum, and is therefore a definite abortifacient drug. This effect has been discussed in considerable detail by Blair Bell and his co-workers in connection with the use of lead in the

treatment of new growths. Dilling found that it also caused tonic contractions of the uterus,[1] and suggested that this might be a factor in the abortions met with in lead workers.

It is commonly taken in the form of pills made from diachylon plaster and there is no doubt that the trophablast may be injured sufficiently to cause the death of the fœtus in doses which do not seriously affect the mother. On the other hand large numbers of cases have been recorded in which definite signs of chronic lead poisoning have supervened before the expulsion of the fœtus. There is of course always a danger of overdosage in any form of self-medication and particularly when drugs are used for the purpose of abortion.

In *R.* v. *Goodall* the prisoner was charged with manslaughter, and with supplying pills for the purpose of procuring abortion. Pills of two kinds, namely diachylon and aloes respectively, were administered. As a result, the woman miscarried, and although she lived for a fortnight after taking the pills, eventually died, with symptoms of intense headache, convulsions, anæmia, etc. On analysis, lead was found in as little as an ounce of the liver. It would appear that a quantity equivalent to 20 grains per day of the diachylon had been taken for several days. The prisoner was sentenced to seven years' penal servitude.

Diachylon Plaster has been subjected to restrictions on its sale which are now those of the First Schedule to the Poisons Rules, 1935, made under the Pharmacy and Poisons Act, 1933. This restriction on its sale has no doubt tended to lessen its use for abortifacient purposes.

Potassium permanganate and Manganese dioxide. The clinical evidence as to the value of these salts as emmenagogues is conflicting, but there is no pharmacological evidence of any action on the uterus. Cases of abortion occurring after the administration of potassium permanganate are recorded, but this result was probably due to the general condition of the patient apart from the drug.

Hormone preparations. Recent progress in the study of the female sex hormones has led to the use of several preparations, natural and synthetic, in the treatment of menstrual irregularity, habitual abortion, and other gynaecological conditions. There is no doubt that a few irresponsible or unscrupulous medical practitioners have been tempted to use certain of these preparations, notably the synthetic oestrins, for the purpose of inducing abortion improperly and illegally. There is little or no evidence of their use outside the medical profession. It is probable, however, that these substances do not have an abortifacient effect on pregnant women, except perhaps in exceedingly large doses, which are sufficient to produce other effects, unpleasant and even dangerous. In this, they resemble most of the other agents possessing an abortifacient reputation.

THE NATURE OF THE EVIDENCE WHICH MAY BE GIVEN WHEN ANY OF THE FOREGOING METHODS ARE ALLEGED TO HAVE BEEN APPLIED

Elsewhere will be found the signs of delivery—they need not be repeated (*vide* pp. 45 *et seq.*). They simply prove (or disprove) the fact of delivery: we must discuss here the medical evidence of how or why delivery occurred.

[1] *B.M.J.*, November 20th, 1926.

Evidence when Disease is alleged as the Cause. This cannot be discussed at full length ; we have given a few of the more important or commoner diseases with which abortion is associated, and the medical evidence consists largely in showing (1) that such disease existed and (2) that there is no evidence, local or general, of violence or drugs having been employed. For the former the medical witness must use his own judgment and professional knowledge when acute or marked disease is in question, but he will have to trust to general information as to previous abortions or vaginal discharges, etc., for much that is valuable ; local examinations may show old para- or perimetritis quite sufficient to account for abortion. Evidence as to drugs may be derived from the symptoms or from analysis of the discharges or of the viscera (*vide* pp. 114, *et seq.* and pp. 125 *et seq*).

Evidence when Violence is alleged. When general violence has been criminally used, it is only the fact that marks of violence are found on a woman who is or was pregnant that can cause suspicion of attempted or completed abortion. The violence can be judged on general principles –the intention with which it was inflicted must be judged by circumstances. There is one point to be emphasised here : if a criminal has resorted to kneeling on the abdomen, much violence may be done internally without leaving any bruises on the skin.

When local violence is alleged, the question of proving that such violence has been applied is very difficult, for, as we have seen, skilled criminal violence may leave no traces whatever.

In these cases a medical opinion should not be based upon the statements either of the woman or of her friends, but upon some distinct and satisfactory medical proofs that mechanical violence has been done to the womb, its contents, or its appendages. Peritonitis i.e., inflammation of the lining-membrane of the abdomen, may arise from a variety of causes. If we assign it to a particular cause, and thus implicate another in a felonious charge, we should do this only upon *medical* facts obtained by an examination of the dead or living body ; we should deal with such cases as if we knew nothing of their history.

In one case, it was suspected that the death of a woman had been caused by attempts made to produce criminal abortion. After three days' illness she was taken in labour and was delivered of a dead child, which was between the sixth and seventh months of uterine age, and she died a few hours afterwards. On an inspection of her body, it was found that the cause of death was peritonitis. She had previously complained of great pain in her abdomen, and peritonitis had developed before she was delivered ; the peritonitis appeared to be the direct cause of the abortion. She admitted having taken some powders to cause miscarriage, and that a person calling himself a medical man had about a week before introduced two instruments into her body, which had caused her great pain. Besides extreme inflammation of the peritoneum, which was the immediate cause of death, the heart, lungs, and stomach were healthy, and the womb presented no appearances excepting those arising from recent delivery ; it was perfectly natural, and free from all marks of injury. There was no injury to the vagina, nor any wound in the peritoneum itself. There was no mark of violence on the body of the child ; in short, this could have sustained no injury, as the membranes surrounding it were not ruptured.

The medical man who examined this case thought that the fatal peritonitis had been caused by the introduction of instruments into the vagina, and that this might occur without leaving after death any

traces of their employment. A speculum used in the ordinary way would not produce peritonitis, and it was alleged in defence that a speculum only had been used. The connection of the peritonitis with the alleged manipulations of the unlicensed practitioner rested more on surmise than on proof. The absence of any bruise, puncture, or laceration affecting the vagina, uterus, or fœtus, with the fact that, whatever may have been the instruments used, the membranes were left entire, rendered it impossible to assign the peritonitis with certainty to the acts of the person who was charged with causing the death of the woman. For anything that appeared to the contrary, he might have used a speculum, and it is well known that this instrument, although frequently introduced into the vagina, does not cause peritonitis. The connection of the peritonitis with instrumental violence, therefore, was not established in this case, and the jury discharged the suspected person. They could not do otherwise, for there was not the slightest *medical* proof that any improper instrument had been introduced into the vagina with felonious intention.

A druggist was charged with using instruments to cause abortion, leading to the death of a woman from peritonitis. He had given to her doses of the tincture of perchloride of iron. The woman was delivered of a dead fœtus at about the fifth month, and died shortly afterwards. There was nothing in the body of the woman or of the fœtus to show that instruments had been used, but it was quite clear that peritonitis was the cause of death. One medical witness thought that an operation had been performed on the body of the woman, but it was admitted that peritonitis might arise from a variety of causes in a woman who had had a miscarriage.

Marks of forceps on the cervix, bruises about the cervix or vaginal walls and small abrasions should be looked for.

The mucous membrane of the vagina, the cervix and cavity of the uterus must be carefully examined for evidence of the passage of an instrument before the parts are manipulated. If such marks are found, it is advisable to have the organ at once preserved in Kaiserling's solution for future examination.

When labour is impeded by a serious disproportion between the size of the head and the calibre of the passages the uterus may be torn (either in its body or in its attachment to the vagina) by its own contractions. If such be alleged to be the cause of such a rupture, there is usually abundant evidence as to the real cause of the damage, for the abortionist using such violence rarely stops at vagina or uterus, but damages other organs as well. The clinical fact of such rupture has a more important bearing when ordinary malpraxis is alleged against a medical man (*vide* Vol. I, pp. 62 *et seq.*).

Marks on the Child. It is obvious that when an instrument is used, injury may possibly be inflicted upon the child. This is more likely to happen with an ignorant person than in skilled attempts, and more likely with sharp pointed instruments than with blunt catheters, etc. If these marks are found, they will be very important evidence. They may not be sufficient to account for the child's death ; but if it can be proved that they have not resulted from accidental causes during gestation, or subsequently to delivery, their presence may furnish strong corroborative evidence of the actual means by which abortion was produced.

Evidence when Drugs are alleged to have been given. A medical witness should be careful in giving evidence as to the properties of drugs. In all cases of alleged criminal abortion by drugs he must consider whether the substance is noxious, and must be prepared to be cross-examined on the ecbolic properties of the substance, as well as on its general action as a poison on the woman or the child.

The medical witness must also bear in mind that the strength of the drug used is of great importance. For example, a decoction of old ergot may have no action whatever.

Noxious Thing. The actual words in use in the statute are "a poison or other noxious thing."

It would appear that it is not in all cases necessary to prove by medical evidence that the substance procured or administered was of a noxious nature. The words of sect. 59 of the Offences against the Person Act, 1861, as to procuring a noxious thing, or any instrument or "thing whatsoever," strictly interpreted, should probably include all substances, noxious and innoxious. In *R.* v. *Wallis* the substances procured by the accused were not noxious, but the jury refused to convict on the ground that he did not administer the drugs; so that the question of noxiousness did not formally arise. It would appear that if a person procured or administered castor-oil or camphor julep with intent to procure miscarriage and in the belief that the substance would produce it, he would be found guilty of the offence. This being so, it would seem that the use of the words "poison" and "noxious thing" in the statute is surplusage, and tends only to cause confusion in the medical evidence.

In Chapter XII will be found a wider discussion and an attempt to define poison and noxious thing with reference to all circumstances. We may here confine ourselves to a brief discussion of the following questions:—

1. *For what alleged symptom was the drug in question administered? Why was the particular drug chosen?*

Women will frequently deceive a medical man with regard to their symptoms in order to induce him to administer drugs, which they hope may have the desired effect, but often enough an abortionist will wilfully misstate symptoms in order to shield himself. Cross-examination should here be able to bring out the motives. A man was, for instance, accused of procuring abortion by administering oil of savin.

The medicinal dose, as an emmenagogue, on the authority of Christison, is from two to five *minims*, and according to Pereira from two to six *drops*. The quantity given by the prisoner, although a full dose, was not greater than these authorities recommend; and his criminality appears to have rested not so much on the dose given, as on the question whether he knew or, as a medical man, had reason to *suspect* that the female for whom he prescribed it was pregnant. No medical authority would recommend oil of savin in full doses for *pregnant* women; and with regard to the existence or non-existence of pregnancy in a special case, medical men are reasonably presumed to have better means of satisfying themselves than non-professional persons. The prisoner's innocence, therefore, rested on the presumption that he implicitly believed what the prosecutrix told him regarding her condition—that he had no reason to *suspect* her pregnancy, and therefore did not hesitate to select and prescribe a medicine which certainly has an evil reputation, and is rarely used by medical practitioners. According to the evidence of the prosecutrix, she informed the prisoner that she had disease of the

heart and liver, and that nothing more was the matter with her. There can be little doubt that the oil was administered with a guilty intention. Every qualified practitioner, acting *bonâ fide*, would undoubtedly satisfy himself that a young woman whose menses were obstructed was *not pregnant* before he prescribed full doses of this oil three times a day, or he would fairly lay himself open to suspicion of criminality. If pregnancy—a frequent cause of obstructed menstruation—were only *suspected*, this would be sufficient to deter a practitioner of common prudence from prescribing, in any dose, a drug which may exert a serious action on the uterine system.

2. *Is the alleged drug known or reputed to have any specific and direct action on the uterus?*

Attention must be drawn to the distinction between known and reputed—the former refers to recognised medical opinion, the latter to hearsay amongst the laity.

Abortion has followed the use of cantharides, caulophyllin, cimicifuga racemose, potassium permanganate, senega, pennyroyal and other essential oils and a variety of purgatives. It is quite possible that any one of these drugs in large doses might cause abortion in a person predisposed to it, but there is no evidence to show that, except in excessive doses, they will cause abortion in a healthy woman. Powerful purgatives are likely to produce abortion through causing congestion of the pelvic organs and possibly from reflex irritation of the uterus. There are a large number of drugs which are reputed to have an abortifacient action but this rests on no scientific foundation. Abortion may occur after the the use of a drug sometimes from its general effect but this does not prove that it has any ecbolic action.

Lead, phosphorus, ergot, rue, and savin all appear to have some abortifacient action.

3. *Is it known to have produced abortion?*

This must be answered in the affirmative of all the drugs we have been considering, or rather, perhaps it is more correct to say that abortion has been known to follow after their administration.

4. *In what doses and with what frequency was it administered?*

Savin and rue are irritant; and they become noxious when given in large doses, or in small doses frequently repeated. Aloes and castor-oil are innocent when taken in small doses; but they acquire noxious or injurious properties when administered frequently, or in large quantity, to a pregnant woman. The small quantity of the substance taken at once does not affect the question, provided the dose be frequently repeated.

In an old case two powders, weighing each one drachm, were prescribed by the accused; one consisted of colocynth, the other of gamboge, and with them was half an ounce of liquid (balsam of copaiba). They were to be mixed together, and a fourth part to be taken four mornings following. A medical witness said, in answer to the question whether such a mixture was noxious or injurious, that each dose would be an active purgative, and might thereby tend to produce abortion. One dose would not be productive of mischief in a healthy country woman, but its frequent repetition might lead to serious consequences in a pregnant woman.

Hence when a purgative or indeed any drug, is known to have been administered to a woman in excessive doses or with excessive frequency, one must ask the question why?

5. *Is the drug in question commonly recognised as an active poison?*

This obviously raises the further question, If so, why was it administered at all? Lead, savin, rue, phosphorus would naturally come under this head. In another old case the accused made the woman take a quantity of *white hellebore*, in powder, for the purpose of procuring abortion. One medical witness said that he considered hellebore to be noxious to the system, but he knew of no case in which it had produced death; and in these circumstances he did not consider himself justified in calling it a poison. Another medical witness stated that, in his opinion, it belonged to the class of poisons. The judge, in summing up, told the jury that *that* was to be regarded as a poisonous drug which, in common parlance, was generally understood and taken to be such; and he thought the medical evidence sufficiently strong to bring hellebore within the meaning of the statute. The jury found the accused guilty alleging that in their belief white hellebore was a poison. It is remarkable that any doubt should have been entertained by a medical practitioner respecting the poisonous properties of white hellebore. It is a powerful vegetable irritant, and has caused death in several instances; yet on this occasion it appears to have been admitted to be *noxious*, but not *poisonous*.

It must, however, never be forgotten, with reference to this crime, that whether the drug would or would not, whether it did or did not, have the intended effect, and whether the woman is injured or not—all these are quite immaterial if only the motive can be reasonably proved to the satisfaction of the jury.

To sum up, no single drug in ordinary doses can be said to be a certain abortifacient. Some drugs, such as cathartics, from their action upon neighbouring organs, may produce abortion. Other drugs, such as cantharides and savin, only produce abortion when given in poisonous doses and by producing poisonous symptoms. Others, such as ergot, undoubtedly act upon the uterus, and if taken in a number of cases will no doubt in a few set up uterine contractions and so induce abortion. Others such as lead lead to the death of the fœtus. The majority of so-called abortifacients are uncertain in their action, and their exact *modus operandi* has yet to be established. It may be said, however, that any pregnant woman taking a reputed emmenagogic or ecbolic drug runs a risk of producing abortion, especially if she has had previous miscarriages. It is nevertheless impossible to say that any given drug in any given dose will produce abortion with certainty, and therefore the **production of legitimate abortion by means of drugs is never practised at the present day.**

Far otherwise is it with abortion-mongers, who fatten on the credulity and folly of women.

In a series of articles the *Lancet* gave many analyses of pills and potions sold as abortifacients. To repeat most of them would be a waste of time, but the following account of *R.* v. *Bedford* may serve as an example.

R. v. *Bedford*. A police officer wrote a letter to the accused representing himself to be "Mrs. E. A. Y.," a woman two months advanced in pregnancy, with the suggestion that the defendant should supply something to procure abortion, and on 18th November he received in reply a cigar-box containing an 8 oz. bottle of

medicine and a box of twenty-four pills and a letter instructing "E. A. Y." to take "two tablespoonfuls of the medicine night and morning," and that "the bottle will require a good shaking before taking it," and "you must take three pills with each dose of medicine."

The following table shows the result of the analysis of the medicine and pills :—

	Total quantity found in 3-oz. bottle by Mr. Orsman.	Quantity in each dose of medicine.	Quantity found in each pill.	Quantity in each dose of pills.	Total at one time for 4 days, night and morning.
Colocynth	67 grs.	8½ grs.	—	—	81 grains
Aloes .	90 grs.	11 grs.	Extractive matter containing both aloes and ergot, 1·7 grains	Extractive matter probably about 5·15 grains: 5·2 grs.	13·5 grs., about
Ergot (really Ergotin)				2·5 grs.	2·5 grs., about
Borax .	77 grs.	91 grs.	—	—	91 grains
Savin .	5 grs.	$\frac{6}{10}$ of a grain	·21 grain	·63 grain, about	1·23 grs., about
Sulphate of Iron	—	—	·96 grain	3 grains, about	3 grains, about
Hellebore .	—	—	1·2 grains	3 grains, about	3 grains, about

On Friday, the 19th of November, the police officer sent a woman A to defendant's. A. represented herself to have missed her courses for about nine weeks, and said she did not want to be bothered with any more children. The defendant supplied her with an 8-oz. bottle of medicine and a box of pills, and also a powder. The result of the analysis of these three things, and of the instructions given by the defendant, would be as follows :—

	Total quantity found in 8-oz. bottle by Mr. Orsman.	Quantity in each dose of medicine.	Quantity found in each pill.	Quantity in each dose of pills.	Total at one time.
Aloes .	90 grs.	11 grs.	Extractive matter containing both aloes and ergot, 1·7 grains	Extractive matter, 3·4 grs., probably containing: 1·7 grs.	12·7 grs.
Ergot (really Ergotin)				1·7 grs.	1·7 grs.
Borax .	77 grs.	9·5 grs.	—	—	9·5 grs.
Oil of Savin	5 grs.	·6 gr.	·21 grain	·41 grain	1·1 grs.
Sulphate of Iron .	—	—	·96 grain	2 grains	2 grs.
Hellebore(?)	—	—	1·2 grains	2·4 grains	2·4 grs.

That is, on Friday night, the 19th, she would take the quantities mentioned in the last column: on Saturday morning, the 20th, she would take similar quantities; on Sunday, the 21st, she would take similar quantities three times

during the day; on Monday morning she would take similar quantities three times during the day; and she would then take the powder at two equal doses. The powder consisted of 58 grains of colocynth.

On the 1st December, the police officer sent a woman B. to the defendant's. B. informed the defendant that she had missed her courses for seven weeks. The defendant gave her a bottle of medicine and a box of pills, and instructed her to take two pills and two tablespoonfuls of the medicine at the same time twice a day. The result of the analysis will be seen in the following table:—

	Total quantity found in 8-oz. bottle by Mr. Orsman.	Quantity in each dose of medicine.	Quantity found in each pill.	Quantity in each dose of pills.	Total at one time for 4 days, night and morning.
Colocynth	67 grs.	8½ grs.	—	—	8½ grs.
Aloes	90 grs.	11 grs.	Extractive matter containing both aloes and ergot, 1.7 grains	Extractive matter 3.4 grs., probably containing 1.7 grs., ab't	12·7 grs., about
Ergot (really Ergotin)				1·7 grs., ab't	1·7 grs., about
Borax	77 grs.	9·5 grs.	—	—	9·5 grs.
Oil of Savin	5 grs.	·6 gr.	·21 grain	·41 grain	1·1 grs.
Sulphate of Iron	—	—	·96 grain	2 grains	2 grs.
Hellebore(?)	—	—	1·2 grains	2.4 grains	2·4 grs.

The police officer subsequently ascertained that the pills were supplied by a Liverpool firm (Sumner Co., of Lord Street, wholesale chemists), and they informed him that the above analysis was practically correct, and that the pills were what were known in the trade as an emmenagogue.

The analyst was the Wigan county borough public analyst.

The defendant advertised herself as Madame Bedford, female specialist and medical herbalist. The jury found her guilty of supplying noxious things, etc., with intent, etc., and she was sentenced to six months' imprisonment with hard labour.

FEIGNED ABORTION

For various motives, into the consideration of which it is unnecessary to enter, a woman may charge another person with having attempted or perpetrated the crime of abortion. Such a charge is not common, because, if untrue, its falsity may be easily demonstrated. A young woman charged a policeman (who, she alleged, had had forcible intercourse with her) with having given her some substance to produce abortion and having subsequently effected this mechanically. She was not examined until nearly two months after the alleged perpetration of the crime, when it was found that there was no reason to believe that she had ever been pregnant. This was a case of feigned abortion. When charges of this serious kind are brought forward they are always open to the greatest suspicion unless made immediately after the alleged attempt, as it is then only that an examination can determine whether they are true or false. If so long delayed, as in this instance, without any satisfactory reason, the assumption is that they are false.

ABORTION OF MONSTERS, MOLES, AND IN EXTRA UTERINE FŒTATION

The law uses the term miscarriage, a popular word, and it intends thereby to mean the discharge of the contents of a gravid uterus, whether such contents be well or ill formed, living or dead, moles, or any other result of conception; the thing is the *intent* with which an operation was done or a drug given.

Again, a person would be equally liable for the attempt whether the fœtus was in the uterus or in the Fallopian tube. The symptoms of extra-uterine pregnancy are similar to those of ordinary pregnancy, and are not to be distinguished from them in the early stages. In an advanced stage the case is different; the symptoms are wholly unlike those of pregnancy, and may wrongly give rise to the suspicion that the woman has died from criminal interference.

A young woman, believed to be some months advanced in pregnancy, died very suddenly soon after taking some medicine prescribed for her. She had enjoyed excellent health, with the exception of being occasionally subject to slight abdominal pains threatening abortion, and to relieve these pains a physician was consulted. It seems that she had aborted on a previous occasion. She was found to be in a state of great depression, but not suffering at the time from any dangerous symptoms. The physician had prescribed a sedative medicine, of which the patient had taken only three doses when she fell into a deep sleep, and in this state she died. The family attributed her death to some mistake in the preparation of the medicine. Upon an inspection of the body, a quantity of blood was found effused in the lower part of the abdomen. This had obviously arisen from the rupture of a tumour, containing an embryo of which the remains were found in the midst of the clots of blood in the pelvis. It appeared to be of only a few weeks' development. The body had been contained in a cyst external to the uterus, which had suddenly given way, and had thus led to fatal hæmorrhage.

It was the suddenness of death soon after taking medicine, without any preceding symptoms of illness or any other obvious cause except the medicine to account for her condition, that gave rise to the inquiry.

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CHAPTER IX

NATURAL BIRTH AND MEDICO-LEGAL QUESTIONS CONNECTED THEREWITH

The Legal Position of the Unborn Child "en ventre sa mere."
Birth Independent of whether Alive or Dead.
- Definition of Birth.
- Sex of Child.
- Exact Time of Birth.
- Cæsarean Section.
- Craniotomy.
- Monsters.
- Plural Births.

Succession to Property.
Live Birth.
- Evidence from Muscular Movements.
- „ „ Warmth and Rigor Mortis.
- „ „ Crying.
- „ „ Respiration: Has this Child Breathed? Change in Shape of Chest.
- „ „ „ Position of Diaphragm.
- „ „ „ The Lungs.
- „ „ „ Stomach and Bowels.
- „ „ Circulation.
- „ „ Contents of Alimentary Canal.
- „ „ The Mode of Birth.
- „ „ The Umbilical Cord.
- „ „ The Skin.

THE UNBORN CHILD

So long as the infant remains in the womb it is said in law to be *en ventre sa mère*: but for many purposes it is legally supposed to have an existence of its own. A child in the womb may be the object of a gift; it may sometimes have a guardian assigned to it; but none of these conditions can take effect unless the child is subsequently born alive. The Roman and the English systems of law apply the same term (*venter*) to the unborn child; when born dead it is called *abortus*, abortion: when alive, *partus infans*, infant.

The maxim of English law, which was derived from the Roman law, is that if any question arises for its benefit, a child *en ventre sa mere* is to be considered as born. This principle was recognised by the House of Lords in *Blasson* v. *Blasson*,[1] but this fiction is applicable only for the purpose of enabling such child to take a benefit to which it would have been entitled if born. A child *en ventre sa mère* at the time of the father's death is entitled to damages.

Medical interest lies only in the establishment of pregnancy subsequently in the nature (sex, shape, etc.) of the products of conception (*vide infra*).

[1] 2 De G. J. & Sm. 665.

BIRTH ALIVE OR DEAD

By the law of England "birth" signifies "the entire delivery of a child," with or without its separation from the body of the mother. (*R.* v. *Crutchley*).[1] The fact that the child had breathed is not a decisive factor, as it may have breathed and yet have died before birth. (*R.* v. *Sellis*).[2] (See "Infanticide.")

On a Saturday evening a woman was taken in labour with her first child. The head and one arm were born two or three minutes before a neighbouring clock struck twelve. There was a cessation of pain for several minutes, during which time the child cried and breathed freely. The rest of the body was not expelled until full five minutes after the same clock had struck twelve. Was the child born on the Saturday or the Sunday?

It is clear that the birth was not completed until the Sunday; the child was still partly within the body of the mother; and the circulation was still maintained through the umbilical vessels. It is elsewhere stated ("Infanticide," *post*) that English judges have decided that no child can be considered *legally* to be born until the *whole* of its body has come *entirely* into the world. In the case under consideration, the child could not, therefore, have been born on the Saturday, because the law does not regard partial birth as entire birth; and respiration and birth are not synonymous terms. If the child had died before its body had been entirely extruded, it could not be said, even medically, that it was born alive; and certainly it could not be considered, according to the present state of the law, to have acquired the rights of a child born living. Whatever apparent injustice may be done by adhering to this rule in respect to civil rights there is no doubt that the evil was of great magnitude in relation to criminal law, for the destruction of partially born children, although alive and healthy, was not legislated for until the passing of the Infant Life Preservation Act, 1929, which made the killing of a child during birth a felony.

On the other hand, some difficulty might arise in civil cases if the bare extrusion of a *part* of the body sufficed for all the legal purposes of *entire* birth. It might become a casuistical question as to how much of a child's body should be in the world in order to constitute legal birth: for there is no reason from the medical point of view, why the extrusion of the head and shoulders should constitute birth any more than the extrusion of a hand or a foot. If it be said that the act of breathing should be combined with a partial extrusion of the body, this would be unjust, because the child is alive—its heart is evidently pulsating, and its blood circulating, as freely before breathing commences as afterwards. Moreover, a child may be born alive, and may live for some time, without breathing (*vide* "Infanticide"); and there is no reason in law why such a child should not be regarded as living, in spite of its not having breathed. In many cases, children have been judicially declared to have been born alive, although they had not breathed; and it is clear from numerous reported instances that a child may manifest life for a certain time without leaving in the lungs any evidence of breathing (see "Atelectasis," "Infanticide"). If, then, proof of breathing is not demanded in cases of entire birth, it could scarcely be required in cases of partial birth.

[1] 7 C. & P. 814.
[2] 7 C. & P. 850.

There can be no birth in the eye of the law unless there is "**complete extrusion of the products of conception outside the maternal genital passage.**"

In connection with birth in general, apart from the question of whether the child be alive or dead, the following matters must be considered:

(*a*) The sex of the child.

(*b*) The actual time of birth.

(*c*) Cæsarean section.

(*a*) **Sex.** The sex of a child must be noted carefully (*vide* Vol. I., pp. 110, *et seq.*, for a full discussion of sex). For purposes of public statistics, it appears that pseudo-hermaphrodites are classed as males; and if it transpires later that they are females, a statutory declaration must be made to that effect; of such cases about three occur yearly. There are no special rules for dealing with double monsters.

(*b*) **Date of Birth.** In courts of law medical evidence has been demanded occasionally of the date of birth in cases in which a period of a few days, hours, or even minutes was required to prove the attainment of majority in connection with questions of responsibility in respect of contracts entered into by minors. Some cases of this kind have been decided by the evidence of the accoucheur himself; others, where the accoucheur is deceased, by the production of his case-books. The strictness and punctuality of some medical practitioners, in making written memoranda of cases attended by them, have led in more than one instance to a satisfactory settlement of such actions; this point has been noticed already under "Age." Proof of date of birth is often of importance in cases of legitimacy. There it is not so much a question of hours and minutes as of days and weeks. The matter has been fully considered under the head of "Legitimacy."

(*c*) **Cæsarean Section.** This operation consists in the removal of the fœtus from the uterus of the mother through an incision made in the abdominal wall. Among the Romans it was decreed by Numa that no pregnant woman should be buried until the fœtus had been removed by Cæsarean section; and the Italian laws also made this operation necessary. In 1491 the first authentic case is recorded of the operation being performed on a living woman.

The only observations which require to be made accurately by the medical practitioner are: (i) the precise moment when the child was extracted; (ii) the question of whether it showed signs of life after extraction; and (iii) if the mother died, the precise moment when death took place. It is possible that cases may arise in which the mother may die under an anæsthetic, and the last point may be difficult to determine and may even give rise to differences of opinion between the surgeon and the anæsthetist, but no rules can be laid down for such cases—each one must be determined on its own merits by the facts observed.

Whereas an operation for the extraction of a child which cannot by any other means be delivered *alive* is fully justifiable, it is obviously not justifiable when the woman and the husband decline, although the obstetrician must place before them the risks of the alternative methods of emptying the uterus. If one of the spouses desires it and the other

objects, a very delicate position may arise, in which case it must be remembered that the woman is the patient and, therefore, is the person whom the physician is under duty to assist to the best of his ability, and whose wishes should, therefore, *within reason*, be acceded to. If the woman refused to undergo the operation and the surgeon performed it at the direction of the husband, an action for damages for assault might follow.

If it has been determined that the operation should be performed, the later, up to full time, it is done the greater is the prospect that the child will survive; and, unless some special circumstances concerning the health of the mother demands its performance earlier, it should be performed as near the 280th day as possible. It is desirable that it should be performed before the onset of labour, before the membranes are ruptured, and before any vaginal examinations have been made.

It is very uncertain how long a child *in utero* may live after the death of the mother. It has been stated that it may survive several hours; but this is not borne out by common experience; at all events no time should be lost if a living child be desired. A case is reported in which fifteen minutes elapsed between the death of the mother and the removal of the child. The child was in a state of asphyxia, but recovered under treatment. The longest period of survival reported is thirty minutes, but five or ten minutes is more usual.[1] It is probable that in most cases the death of the child is contemporaneous with that of the mother.

It has happened not infrequently that in ordinary parturition a child is born alive (and survives to adult life) after the death of the mother.

Craniotomy. This means that it is necessary to destroy the child to effect delivery, which otherwise could not take place without leading (probably) to the death of the woman. This operation is not likely to give rise to any medico-legal question, except in a case in which the life of the child has not been completely destroyed before entire delivery. Craniotomy, as the name implies, consists in cutting through the cranium and destroying or removing the brain of the child. In some circumstances there may be movements of the limbs or body after delivery.

The existence of these movements, properly attested by the accoucheur, might furnish important evidence in cases of succession to property. It would be for the court to decide, upon medical evidence, whether the child had in fact been born alive.

If something more violent than puncture be performed on the child's head (**craniectomy, cephalotripsy,** etc.) no question of live birth can arise.

Monsters. Although legal questions connected with monstrous births do not occur often, a medical witness ought to be acquainted with certain facts respecting them. The law of England has given no precise definition of a *monster*. According to Lord Chief Justice Coke, it is a being "which hath not the shape of mankind." A mere deformity in any part of the body, such as supernumerary fingers or toes, twisted or deformed limbs, will not constitute a monster in law, provided the being still has "*human shape*." From Coke's description, it is obvious that the law will be guided in its decision by the description of the monstrous birth

[1] Rosin, I. B. *Lancet* 1; 208, 1927.

given by a medical witness. It is not for a medical witness to decide whether the being was or was not a monster—the court will draw its own inference from the description given. Various classifications of monsters have been made, but these are of no assistance whatever to a medical witness, because each case must be decided by the peculiarities attending it ; and his duty will be not to state the class and order of the monster, but merely to indicate in what respects it differs from a normal human being.

Malpositions, transpositions, or defects of the internal organs of any of the cavities, do not constitute monstrous births within the meaning of English law. The legal question relates only to *external* shape, not to *internal* conformation. Many internally malformed persons live to a great age ; and often malposition and defects are discovered only after death. One test of monstrosity has been based on the viability of offspring. According to some authorities, a monster implies such a malformed being that the child would be pronounced non-viable, *i.e.*, incapable of continuing to live after it was born. The English law does not regard *internal* monstrosity as a bar to civil rights. It is clear from the decisions in *Brock* v. *Kellock*,[1] and in *Jones* v. *Ricketts*,[2] that the simple question in English law is, not whether a child is or is not "*viable*," but whether it has manifested any distinct sign of life after it was entirely born. No person is justified in destroying a monster at birth.

Plural Births. We are not aware of any case on record in which the evidence of a medical man has been required on this subject. It is a simple question of primogeniture. Such questions are usually determined by means of evidence in the form of depositions or declarations of relatives or servants who were present at the birth. In the absence of eyewitnesses, the question of priority of birth must be a matter of conjecture. It cannot be determined by the size of the child. Women may have two, three, four, or five children at a birth. Twins are comparatively frequent, but triplets and quadruplets are very rare.

It is stated that in the Museum of the Royal College of Surgeons of England "there is a large bottle containing five young ladies and gentlemen, all brought forth at one birth, and destroyed by an accident."

In a case of plural births the accoucheur should pay particular attention to the order of their occurrence, and should note carefully whether any or all of them manifested signs of life after birth. In case of twins or triplet males, a practitioner would find himself much embarrassed, after a lapse of time, to state positively which was born first, unless there were some personal peculiarity or deformity which would enable him at once to stamp the identity of a child.

SUCCESSION TO PROPERTY

By the Administration of Estates Act, 1925, which came into operation on January 1st, 1926, the old law as to inheritance and succession to property in England (for example, heirship and tenancy by courtesy) was abolished : and there has been substituted therefor a simple code for the devolution of all forms of property upon the death of a person intestate.

[1] 3 Giff. 58.
[2] 31 L. J. Ch. 753.

LIVE BIRTH

A child is *live-born* according to the law of England when, after complete extrusion from the maternal parts, it exhibits some sign of vitality, such as for example, activity of the heart, breathing, movement of the limbs, crying, etc. Where respiration is not fully established, it is essential for a person who was present at the delivery to give evidence of the complete birth, as well as of the subsequent exhibition of a sign of life.

It was held in *R.* v. *Poulton*[1] that for a child to be born alive, the whole body of the child must have been brought alive in the world. It is not sufficient that the child had breathed in the progress of the birth.

In *R.* v. *Handley*,[2] it was held that a child is born alive when, breathing and living by reason of breathing and living through its own lungs alone, it exists as a live child without deriving any of its living, or power of living, through any connexion with its mother.

Still-births. By the Births and Deaths Registration Act, 1926, a still-born child must be registered in the form given in the First Schedule to the Act. By sect. 12 of the Act a " still-born " child is one which has issued forth from its mother after the twenty-eighth week of pregnancy and which did not, at any time after being completely expelled from its mother, breathe or show any other signs of life.

By the Population (Statistics) Act, 1938, the person who gives information under the Registration Acts of a still-birth must state (*inter alia*) the age of the mother and the date of the marriage.

PHYSIOLOGICAL SIGNS OF LIVE BIRTH

For the purposes of civil and of criminal law it is necessary to consider the physiological signs of live birth, and to discuss fully the nature and value of the deductions which may legitimately be drawn from them.

In order to facilitate reference, the following table is given of the sources whence evidence of life may be drawn :—

1. Breathing.
   - (*a*) Shape of chest.
   - (*b*) Position of diaphragm.
   - (*c*) Air in the stomach or bowel.
   - (*d*) Air in the lungs.
2. Crying.
3. Muscular movements.
4. Circulation and heart-beat.
5. Contents (other than air) of gut.
6. Umbilical cord.
7. Skin.

BREATHING

Whilst in the uterus the fœtus derives its oxygen from its mother's blood, but immediately after birth from the stimulus of the external atmosphere or from interruption of the placental circulation the child attempts to breathe. This causes important and permanent changes

[1] 5 C. & P. 329.
[2] 13 Cox, C. C. 79.

in the lungs the extent of which depends on the completeness or otherwise of respiration. Evidence of respiration is most important in deciding whether a child has been born alive.

(*a*) **Evidence from the Shape of the Chest.** It is said that before respiration the chest is flattened, while after that process it is arched in front. The diameters of the cavity have been measured and certain comparisons instituted, but these experiments have been attended with no practical results.

(*b*) **Evidence from the Position of the Diaphragm.** The position of the diaphragm should be determined by passing the fingers up into its dome through the abdominal incision before the chest is opened. In children who have not respired the diaphragm rises in the chest to the level of the third, fourth, or fifth rib ; after respiration it sinks a good deal and rises the only to fifth, sixth, or seventh rib. On opening the chest the degree to which the diaphragm is covered by the lungs should be noted, for the more the lungs are filled with air the greater will be this degree.

These small points are, however, merely corroborative details in estimating the value of the hydrostatic test results and need not be further discussed.

(*c*) **Evidence from Air in Stomach and Bowel.** To perform the stomach-bowel test, a ligature is placed round the middle of the œsophagus, and it is dissected out with the stomach attached (taking care not to incise the viscus by accident). The dissection is continued to the lower end of the duodenum, a second ligature is placed round this, and the piece of the alimentary canal between the two ligatures removed *en masse* and placed in water. If it sinks there is no air in the viscus, if it floats there is air in it. Air has arrived there either in the process of breathing or swallowing, hence the object of this test is to corroborate the hydrostatic lung test. Thus if the piece of viscus removed sinks in water it is suggestive of want of active respiratory efforts on the part of the child. If, on the other hand, it floats, this indicates (i) natural respiration or (ii) artificial respiration or (iii) gases of decomposition. If evidence of the last two be wanting, we then get corroboration of the hydrostatic lung test. It may be noted that the further down the alimentary canal to which air has passed the stronger is the presumption of respiration. If the indications of lung and bowel tests are opposed, the lung test is the more trustworthy.

There is no reason to doubt these facts, and the deduction concerning breathing which may be made from them. No definite conclusions can be drawn from them as to whether the child was born alive in the legal sense.

(*d*) **Evidence from the Lungs.** The cavity of the chest in a newborn child may be conveniently laid open in a manner somewhat different from that in ordinary use, as follows : —After reflecting the soft tissues from the front of the chest in the ordinary way, the clavicles and the ribs should be cut through and the whole flap of tissue thus formed (of ribs, clavicles, and sternum) should be turned downwards without separating the diaphragm from its attachments.

The thymus gland, as large as the heart, occupies the upper and middle portions of the cavity; the heart within its membrane is situated in the lower and middle portion, and is rather inclined to the left side. The lungs are placed quite in the back part of the chest, so as often to give the impression that they are absent. In some instances they project slightly forwards by their anterior margins; but in no instance, unless congested, infiltrated, or otherwise diseased, do they cover and conceal the heart. The thymus gland is sometimes of a pale fawn, at others of a deep livid colour; but there is no appreciable difference in this organ in new-born children, before or after the performance of respiration.

We have now to see what changes are produced in the chest, or rather in the lungs, as the result of respiration; they may be thus tabulated for completeness, but the importance of the changes is a varying quantity.

Synopsis of Points in the Lungs

Not Breathed	Breathed
1. Volume small, tucked up behind the heart.	1. Volume larger, cover the heart to some extent.
2. Margins sharp, mass solid.	2. Margins rounded, mass spongy.
3. Colour of chocolate, and uniformly so all over.	3. Lighter in colour and mottled; great stress is laid by some experts on this mottling or unevenly coloured appearance.
4. Vesicles not seen on the surface.	4. Vesicles are visible almost to the naked eye.
5. The lung tissue does not crepitate under the finger on squeezing.	5. The tissue crepitates on squeezing.
6. If on squeezing under water any bubbles of gas escape, these are large and uneven in size and due to gases of decomposition.	6. On squeezing under water the bubbles produced are small and uniform in size.
7. Very little blood can be squeezed out. This blood is not frothy, unless it is decomposed.	7. Full of blood, which is frothy on squeezing, though not decomposed.
8. Weight less.	8. Weight greater
9. Sink in water.	9. Float in water.

1. **Volume of the Lungs.** The difference in the relative situation of the lungs before and after respiration depends entirely upon the increased volume or dilatation of the organs, arising from the introduction of air. *Before respiration*, the lungs are in general scarcely visible, unless forcibly drawn forwards in the chest. When it has been perfectly accomplished, the volume is so much increased, that the pericardium is almost concealed by them. Respiration must, however, have been fully performed in order that this condition should exist to the extent described; but the lungs may acquire a considerable volume in a healthy

and vigorous child from only two or three respirations. A child may also live for one or two days, and the volume of the organs be but little altered.

2. **Margins of the Lungs.** These alter in sharpness with the expansion of the lungs with air, becoming quite rounded when complete expansion takes place.

3. **Colour of the Lungs.** The colour of the lungs *before respiration* is brown-red, bluish, or deep violet; but it is subject to variation. It is important to remark, that a very short exposure to air will materially brighten the colour of the lungs in the parts exposed, so that it should be observed and recorded immediately on opening the chest. *After respiration*, the lungs acquire a light red hue in proportion to the degree in which the process has been performed. If imperfectly established they will be mottled, generally about the anterior surfaces and margins; the patches of light red being intermixed with the livid fœtal hue, very much the same as the collapse observed in ordinary *post-mortem* work, and being slightly raised, as if by distension, above the general surface of the organs. The light red tint changes, after a short exposure to air, to a bright scarlet.

Artificial introduction of air by a tracheal tube or otherwise, in the attempt to resuscitate a still-born child, is attended with the same physical change, both in colour and volume, as has been observed in the course of numerous experiments.

The red colour depends on oxidation of the hæmoglobin of the blood. It is a simple chemical phenomenon, and as such is independent of the means whereby the oxygen got there.

4. **Expansion of Air Vesicles.** On the right lung (more commonly), and especially on the edges and concave surface of its upper lobe, the first appearances of respiration may be visible, even when the rest of the lungs retain their fœtal condition. Here it is that the highly characteristic expanded air-cells are first visible. These, if the lungs are fresh and full of blood, take the form of bright vermilion spots; but if the lungs contain less blood, or are examined some days after death, the spots are of a lighter tint. The form and arrangement of these cells are also characteristic; they are angular, and are not perceptibly raised above the surface of the lung. They may be either irregularly grouped, or arranged in sets of four or five; and their outline is distinctly polygonal. They are best seen with the naked eye, or at most with a lens of low power. Their form, their immobility when the finger is passed over the surface of the lung, their colour, and the fact that they are not raised above the surface of the lung, renders it impossible to mistake these cells for the minute bullæ of putrefaction, melanotic spots, or minute ecchymoses, if ordinary care be exercised. Some expansion of air-cells may be brought about by artificial respiration.

In any cases of doubt, the condition of the air vesicles should be verified by the examination of sections of the lung under the microscope.

5. **Consistency of the Lungs.** The lungs, *before respiration*, feel like the liver, or any of the other soft but solid organs of the body. They are under the finger, and their substance may be lacerated by moderate compression. *After respiration* has been fully performed, there is a

distinct sensation of what is termed crepitation on compressing them, *i.e.*, air is felt within them. This condition of the organs will, of course, depend on the degree to which respiration has been carried. The lungs of children that have lived for a considerable time after birth will sometimes give no feeling of crepitation under the finger. Lungs of this kind usually present the other fœtal characters : thus they are small and of a livid colour. There are, however, cases in which the lungs may have the light red colour of respiration, and be actually much dilated in appearance, yet no feeling of crepitation will be perceptible on pressure. This feature therefore is by no means a necessary accompaniment of the other two. Crepitation furnishes presumptive evidence of respiration ; but it may be met with in lungs which are putrefied, or which have received air by artificial inflation. The features here described are seldom found in the lungs of children born prematurely, although such children may have lived some time after birth ; they depend on respiration, and in the exceptional cases referred to this process is only slowly and imperfectly established. Independently of the feeling conveyed by the presence of air, a section of the lungs, examined by the microscope, will enable the examiner to ascertain whether air has or has not penetrated into them ; in the former condition air-cells will be visible, but not in the latter.

6. **Gas in the Lungs.** If there can be appreciated any feeling of gas being in the lungs they must be removed and **squeezed under water.** On squeezing a piece of healthy respired lung (infantile or adult) under water, it will be at once noticed that the bubbles of air are all small and, speaking broadly, of the same size ; whereas when decomposition is the cause of the gases being present the bubbles are uneven in size and they escape irregularly. In decomposition of the lungs it is also possible to see the air beneath the pleura and to move it from place to place in that situation by stroking with the finger or other instrument. It is thus easily possible by this means to distinguish the gases of putrefaction from those of respiration (*vide* also below, under " Hydrostatic Test ").

7. **Blood in the Lungs.** The pulmonary vessels contain blood, both in the child which has and in that which has not breathed, but the vessels contain more blood after respiration than before. The excess of blood after the establishment of respiration, however, is distributed throughout the smaller vessels of the lungs : it does not remain in the large trunks. The state of the larger pulmonary vessels, therefore, furnishes no evidence either of respiration or the contrary, but the blood which escapes on pressure from lungs which have breathed is frothy, a point of very great importance if the source of the gases which make it frothy is known. The blood may be found coagulated or not, and there is no difference in this condition, whether the child is born living or dead.

It is probable that the amount of blood in the lungs is different in the cases of natural respiration from that observed when only artificial respiration has been performed.

8. **Weight of the Lungs, absolute and relative to Body Weight.** Inasmuch as these tests have been put forth by medical jurists as tests of respiration, they must be dealt with.

In order to determine the weight of the lungs, these organs should be carefully separated by dissection from the heart and thymus gland, and removed with the windpipe and bronchi attached. Previously to their removal, ligatures should be placed on the pulmonary vessels, so that no blood may escape from the lungs. They should now be weighed, and the weight accurately noted. In taking this weight it does not appear necessary to make any distinction founded on the sex of the child, or on the difference of weight in the two lungs; the only exception would be, perhaps, in relation to twin children imperfectly developed. The average weight *before respiration*, derived from nine cases, was found to be 649 grains. According to Traill, the weight varies from 430 to 600 grains. It is of importance in taking the weight of these organs to observe whether the child is at or near maturity, and whether it is of or about *the average size and weight*: owing to a neglect of this rule, it is highly probable that comparisons have been made of the absolute weight of the lungs in children of different ages which a full statement of the facts would not have justified. If it be small and immature, or unusually large, the lungs will weigh either less or more than the average. The average weight of the lungs *after respiration*, derived from three cases, was 927 grains. There does not appear to be any strict normal relation between the weights of the body and lungs in new-born children; it is certain that in the bodies of children of unusual weight the lungs will be found much heavier than the average, whether the child has breathed or not. The body may vary from six to eighteen pounds; the lungs in these circumstances will also differ in weight.

The healthy lungs of mature new-born children become heavier after respiration, and according to its degree; and where a deviation from this rule is observed it may probably be explained by the circumstance that the lungs of an immature have been compared with those of a mature child—the lungs of an undeveloped twin with those of one not a twin—or the lungs of one which has breathed imperfectly with those of another in which respiration has become well established. In this respect the extensive tables drawn up by Lecieux are liable to lead to erroneous inferences, relative to the effect of respiration on the absolute weight of the lungs. The weights of the organs are noted, but the *degree* to which respiration had been performed is so loosely stated as to allow of no fair inference of the effect of this process upon the weight. The time which the children survived is stated; but this, it is very well known, furnishes no criterion of the degree to which respiration has been carried. Again, we are not informed whether due care was taken to ascertain if the lungs were healthy or diseased. The following table of the weight of the lungs, in four cases from the author's own observation, will show how much the organs are liable to vary in weight after birth, according to the *degree* of respiration:

Case 1.	Born dead	Weight, 678 grs.	
„ 2.	Lived 6 hours	„ 774	„
„ 3.	Lived 24 hours	„ 675	„
„ 4.	Lived 8 or 9 days	„ 861	„

Relying upon a table of this kind only, without comparing the other characters of the lungs with the weight, it might be inferred that the organs would weigh less in a child which had survived its birth twenty-four hours than in another which had been born dead, and that there would be very little difference in the weight, whether the child lived six hours or nine days; but when it is stated that in Case 3 the lungs had every fœtal character possessed by those in Case 1, and that in Case 4 respiration had been obviously very imperfectly performed, the difficulty is removed. Such cases should rather be compared with the lungs in the fœtal than in the respired state. They merely show what is very well known to and admitted by all medical jurists, that there are some instances in which the fact of respiration cannot be determined by the application of the static or any other test to the lungs.

9. **The Hydrostatic Test.** The specific gravity of the lungs is greater before than after respiration; for although the organs become absolutely heavier by the establishment of the process, this is owing, not to the air, but to the additional quantity of blood received into them. The air thus received increases the volume of the lungs so as to more than counteract the additional weight derived from the blood, and thus to

diminish their specific gravity. In these circumstances they readily float in water. The specific gravity of the lungs before respiration, *i.e.*, in the fœtal condition, varies from 1·04 to 1·05, *i.e.*, they are about one-twentieth part heavier than their bulk of water. After respiration, the specific gravity of the lungs was found in one experiment to be 0·94, *i.e.*, the organs were about one-seventeenth part lighter than their bulk of water. The introduction of a small quantity of air will render these organs buoyant in water, and an alteration in the volume of the lungs sufficient for this purpose would not be perceptible to the eye. It will be understood that the specific gravity of the intrinsic substance of the lungs is unchanged ; the organs are rendered apparently lighter by the air contained in their cells. Hence it follows that the diminution of specific gravity will take place whether the air be derived from respiration, artificial inflation, or putrefaction. It is on this property of the lungs that the application of what is termed the *hydrostatic test*, or the *docimasia pulmonaris*, is founded.

How to Perform the Test

The mode of performing the experiment is extremely simple. Having removed the lungs from the chest, they should be placed, still connected with the trachea and bronchi, in a large vessel of water. If they sink it should be noted whether the sinking takes place rapidly or slowly. If they sink when thus united, the two lungs should be tried separately, for it is sometimes found that one, commonly the right, will float, while the other will sink. Supposing that both lungs sink, it will then be proper to divide each into twelve or fifteen pieces and place these pieces separately in water. On the other hand, the organs when placed in water may float ; it should then be noticed whether they float high above the surface or at or below the level of the water. Sometimes they indifferently float or sink. It will now be proper to separate the lungs, and determine whether the buoyancy is due to one or both. Each lung should be divided, as before, into fifteen or twenty pieces, and all the pieces thrown into the water. The pieces which floated and those which only sank gradually must now be collected, placed in a cloth and squeezed firmly, after which they must again be thrown into water and their behaviour noted : some which previously floated may now sink, others may sink more rapidly, or all may float. Some have recommended that the lungs should be placed in water with the heart and thymus gland attached ; but there appears to be no good reason for this, since it is as easy to form an opinion of the degree of buoyancy possessed by the lungs, from the readiness with which they float, as by observing whether or not they have the power of supporting these two organs. We have now at any rate a complete record of the behaviour in water of both lungs, of each lung, and of every piece of each lung.

Value of the Facts ascertained as above from the Lungs, in deciding whether the Child had Breathed

(i.) **The Volume of the Lungs.** The changes in volume caused by respiration take place rapidly, if they take place at all, and complete expansion has been observed when the child has been known to have taken only a few breaths before death. Sometimes, *per contra*, they

are found in their fœtal condition, though the child has been known to have survived birth many hours. We may meet then with every variety in the appearance between the two extremes : the act of breathing often requires a considerable time in order to be *fully* established, especially in those children which are of a weakly constitution or prematurely born. Hence the lungs will be found to occupy their respective cavities to a greater or less extent, and to cover the diaphragm and pericardium more or less, not according to the time that the child has lived, but according to the perfection with which the process of respiration has been performed. Thus, although, as a general rule, the lungs are more perfectly filled with air in proportion to the time during which a child has survived its birth, yet this is open to numerous exceptions.

(ii.) **Margins of the Lung.** These require no discussion ; we need only mention that for some reason—possibly want of lateral support, as in emphysema in adults—the margins are frequently rounded and distended, when large portions of the lung are still fœtal. Hence it is from the margins that those pieces are likely to come which float when we find the "hydrostatic test," giving us intermediate results, a point which might be noted.

(iii.) **Colour of Lung.** A red as opposed to a bluish purple colour indicates oxidised hæmoglobin, and therefore penetration of air, hence the more uniform the red colour the more perfectly has air penetrated ; the less uniform, the less perfectly has air penetrated. Taylor knew a case in which a child lived twenty-four hours, breathing feebly, and yet its lungs on examination were precisely the colour of fœtal lungs. It will be found that the red parts float, the blue ones sink, in the "hydrostatic test," to which we must again refer.

(iv.) **Visibility of Air Vesicles.** Precisely the same arguments apply as to colour ; the vesicles will be evident in the red parts and not in the blue ; evident in those that float, not evident in those that sink.

(v.) **Crepitation on Squeezing.** Another minor point, but one about which there is this to be said, the manipulation of the lungs necessary to its exhibition is apt to drive air from a crepitant air containing piece into one that was fœtal, hence careless manipulation may lead one to think that penetration of air has been more complete than is really the case. Care should then be taken not to use this test more than can be helped before resorting to the hydrostatic test.

(vi.) **Bubbles of Air from Lungs squeezed under Water.** Care is needed to note the size and unevenness of the bubbles that we may not mistake breathed air for the gases of decomposition ; beyond this the test is merely subordinate to and corroborative of the "hydrostatic test."

(vii.) **Blood in the Lungs.** It is desirable to indicate the difference in result between sucking air into the chest by active expansion of its cavity and forcing air into the chest, causing a passive expansion of its cavity. The former encourages circulation of blood through, as well as air entry into, the lung, by reducing the pressure to which the outer surface of the lung is exposed inside the unexpanded chest : the latter has no tendency to reduce this pressure, but merely increases the counter-pressure in the air tubes, and thus has a tendency to prevent circulation of blood through the lungs.

It is thus possible to conceive that we might get indications, from observing the amount of blood in the substance of the lung, as to the means used to stimulate respiration, differentiating between the skilled efforts of the accoucheur and the unskilled efforts of a mother.

Evidence thus derived might influence our opinion of the results obtained from the "hydrostatic test" and suggest artificial respiration.

(viii.) **Weight of the Lungs.** The facts ascertained are so variable that no definite conclusions can be drawn from this test. The lungs of a child which has not breathed are sometimes found to be heavier than those of another child which has breathed, therefore the absolute weight is of little value in proving the fact of breathing.

As to the relative weights of lungs before and after respiration, there can be no question but that respiration increases the weight of a lung; but the lungs before us are in one condition or the other (as proved by other means), and they cannot be made to alter into the reverse condition, so that it is beyond our power to say how much they may have gained by respiration, or how much they would lose by a return to a fœtal condition. Hence we can only compare them with an average standard, and a comparison with the body weight of the child.

(ix.) **The Hydrostatic Test.** This is a very old test, and to it all the preceding observations on the lung are merely a preliminary (one might say) of descriptive appearances rather than tests. The results of the test may be three in number.

A. The lungs and every piece may float.

B. The lungs may just sink or just float, but of the pieces some float and some sink.

C. The lungs and every piece may sink.

A. **The Lungs and every piece thereof float freely, even after pressure.** This proves that air has penetrated freely to all parts of the lung: this penetration can only be due to (*a*) respiratory acts of the child; (*b*) artificial respiration by some one else; (*c*) putrefaction.

The last two alternatives are discussed below. We must here make a few remarks on the first alternative.

It is natural to suppose that complete expansion of the lung indicates prolonged respiration, and therefore some considerable duration of life leading to a presumption that such duration was too great for complete birth not to have taken place. It can be assumed that complete expansion of the lung is not possible whilst the chest of the child is compressed in the maternal parts, but there is of course a possibility that breathing may occur before the whole of the body is extruded.

In one instance Taylor found it impossible to expel the air when the child had lived to make no more than one or two respirations, and had died before it was actually born. On this occasion it was found necessary, in order to effect delivery, to destroy the child while its head was presenting. It lived, however, a sufficient time after the protrusion of its head, with the greater part of the brain destroyed, to cry loudly for an instant. The general appearance of the body showed that it had attained to the full period of gestation. On opening the chest, the lungs were seen projecting slightly forwards over the sides of the pericardium. They were of a light red colour, but not crepitant under the finger. They had the external physical characters which these organs are known to acquire on the first establishment of

respiration ; but the absence of crepitation proved that the air-cells were not completely filled. The colour of the external surface was throughout uniform, a circumstance which the author never witnessed in lungs that had been artificially inflated, except when the inflation had been carried to its fullest extent out of the body. Then, however, there is commonly distinct crepitation. When removed and placed on water, the lungs floated freely ; and, on being separated, both appeared equally buoyant. Each lung was next divided into sixteen pieces and every piece floated. In dividing them, it was observed that the colour was uniform throughout their substance, but there was no sense of crepitation under the knife ; and the cells in which the air was diffused could not be seen. The pieces were then subjected to forcible compression for some time in a folded cloth. The cloth was ruptured by the force employed ; yet, on removing the pieces, and placing them on water, they all continued to float. A portion of air had, undoubtedly, been forced out, but not sufficient to deprive any of them entirely of their buoyancy. The compression was carried to the farthest possible limit consistently with the preservation of the structure of the lungs.

The position then is, that although there may occur cases in which the signs of full respiration may justify an opinion of live birth, such opinion will have to be fortified by other facts in addition before it can amount to conclusive proof.

B. **The Lungs just float or sink, but some pieces float and others sink.** This condition is obviously due to less complete penetration of air and proves less vigorous respiration.

C. **The Lungs and every piece sink.** Obviously no permanent expansion has taken place suggesting no respiration, but this may not be accurate, for though the child may have made efforts at respiration, the lungs or fragments may sink from intra-uterine disease of the lungs, or persistent simple atelectasis.

We should also remember that though the child has made no perceptible efforts to breathe, the lungs or fragments may float from putrefaction or artificial respiration.

The Lungs or Fragments may Sink though the Child has made Efforts at Respiration

(*a*) **From Disease.** Syphilitic disease of the lungs is known in new-born babies, and pneumonic or other consolidation is occasionally found ; or, the lungs may be œdematous from morbid changes in the circulation. None of these, however, need cause insuperable difficulty, for they can be diagnosed by microscopic examination.

(*b*) **From Persistent Atelectasis.** It is remarkable that life should continue for many hours, sometimes even for days, in such a condition ; but, nevertheless, the occasional existence of this state of the organs (either as a whole or in patches) in a living child is placed beyond all dispute. Nay, more, it has been found that it is not necessary that the whole of the lungs should have received air in order that a child should continue to live for months after its birth. It is only necessary to quote three such cases, one twelve hours, another five weeks, and a third six months.

Donders made a *post-mortem* examination of the body of a child for one of his lectures on Forensic Medicine. The lungs were of a uniformly brown colour, placed rather on the side of the chest, with their edges superiorly. They both sank in water ; the right was readily inflated ; the left was cut into pieces, during which process no crepitation was heard or felt, and each piece sank in water. A

knife passed with light pressure over the section expressed only a little reddish-coloured fluid. The bladder was empty. There was no meconium in the large intestines. There was no food in the stomach. The conclusions were—(1) an immature child of about seven months, still-born, which did not remain in the uterus, for more than a short time after death : (2) only a short time dead.

The error of this conclusion was subsequently demonstrated by the ascertained facts of the case. The child at its birth gave but slight signs of life, but on the employment of the ordinary means it soon began to cry in the usual way. For some hours it lay quietly moaning. In the evening it was of a bluish colour but became more lively on the application of warmth. It soon grew cold and rigid and died *twelve hours* after its birth.

A child, aged five weeks, died suddenly, and its death was attributed to an opiate, although the circumstances rendered it highly improbable that the child had died from poison. The body was in good condition. The lungs were found lying at the back part of the chest, inelastic, and presenting no crepitation in any part. They had the usual appearance of the unexpanded lungs of the fœtus. They weighed 1,080 grains. They sank in water, and when divided into many pieces no portion of them floated. It was difficult to inflate them, and the portions inflated readily lost the air by compression and sank. The microscope showed an absence of cellular structure. It is surprising how the child could have lived so many weeks with this state of the lungs ; and it is obvious that in such circumstances a very slight obstruction to respiration would suffice to account for its sudden death.

A child aged six months had been, it was supposed, destroyed by suffocation. Upon opening the chest the viscera were found healthy ; but the whole of the inferior lobe of the right lung was, so far as regarded colour, density, and structure, precisely like the lungs of a fœtus, no air having ever penetrated into it. It had grown in size but never functioned.

Meckel relates two instances in which the lungs sank in water, but the women respectively confessed that they had destroyed their children.

The cases given above demontrate the fallacy of those medical opinions which have been given by some experts in civil cases, involving questions connected with live birth, inheritance, etc. Looking to the condition of the lungs alone, it is obvious that many children would be pronounced dead who were not only living when born, but had survived their birth many hours. Such a life, although not indicated by those changes in the lungs which are brought about by active respiration, must still be called extra-uterine.

Mashka and others deny that air enters the lungs at all in such cases : the passage of air along the treachea and bronchi is regarded as sufficient to account for the signs manifested during life. Others accept the theory first propounded by Simon Thomas—that in feeble infants the respiratory movements may gradually subside in such a way that the passive elasticity of the lung-tissue, at every respiration, drives out more air than is drawn in at the inspiration ; in this way the lungs, after having breathed, gradually return to the fœtal condition. As the result of experimental investigation Ungar states that the air which has entered the *lungs may be entirely absorbed* after respiration has ceased by the blood circulating through them.

Possibly also the following may explain matters in some cases. A healthy, full-grown child, recently born, may make an attempt at inspiration, but the closure of the larynx from spasm, or some irritant such as the vaginal discharges, meconium, etc., may impede the entry of air into the lungs. The chest in this case is arched, the head thrown back, and there is a convulsive rigidity of the muscular system ; the tongue is firmly retracted, especially at its base. Unless the finger of

the accoucheur is passed quickly down to the base of the tongue, and the epiglottis raised by pressing it forwards, the child would never inspire, although it might have a perfect capacity to breathe. Braxton Hicks met with a case of this kind: the air entered the lungs immediately after the above operation, and the child breathed and lived.

Any discussion on these explanations would be useless and out of place here when the facts themselves, with which alone a medical jurist is concerned, are attested by so many independent witnesses.

The Lungs or Fragments may float though the Child has not made Efforts at Breathing

(*a*) **From Putrefaction.** The lungs of a still-born child, when allowed to remain in the thorax, are slow in undergoing putrefaction; but they sooner or later acquire sufficient gas to render them buoyant in water. This form of gaseous putrefaction may even take place in the lungs of a child which has died in the womb.

When the lungs are putrefied, this will be determined, as a rule, by putrefaction having extended throughout all the soft parts of the body. The organs, according to the degree of putrefaction, will be found soft, of a dark green or brown colour, and of a highly offensive odour; and the serous membrane covering the surface will be raised in large visible bladders, from which the air may be forced out by very moderate compression. In the same conditions, gaseous putrefaction takes place more rapidly in the other viscera of a new-born child than in the lungs. We should, therefore, examine the general conditions of these organs and the body. The distension of the lungs with gas from putrefaction cannot be easily overlooked nor mistaken for the air of respiration. A case may possibly occur wherein the characters presented by the lungs will be such as to create some doubt whether the buoyancy of the organs is due to putrefaction or respiration; or, what is not unusual, whether the putrefied lungs may not also have undergone the changes produced by respiration.

The hydrostatic test may enable the examiner to distinguish putrefaction from respiration as follows:—

If a piece of decomposing lung be firmly squeezed, as mentioned in the details of the test, the gas will be easily forced out, because, owing to decomposition, passage in any direction through the tissues is easy, and the lung will then sink. This is in contrast to the behaviour of a piece of respired lung (not decomposed), for in this case the air is only able to travel along the natural passages, and it is practically impossible, in applying moderate or even severe pressure (in the ordinary way), to avoid blocking some of the tubes in the middle, and so retaining some of the air in other distal parts; hence, short of pressure so severe as to destroy their texture altogether, the lungs will float by reason of this imprisoned air. When decomposition has advanced, the differences will disappear because the respired air in the tubes can find, equally with the gases of decomposition, a free passage in any direction.

Other facts, including the examination of microscopic sections of the lung, may serve to remove any doubt.

It may be that the medical witness cannot obtain satisfactory evidence from experiments on lungs in such a condition. He should then at once

abandon the case, and declare that in regard to the question of respiration, medical evidence cannot establish either the affirmative or the negative. In a case of poisoning, the appearances after death in the viscera may be entirely destroyed by putrefaction; but no practitioner would think of looking for proofs when the circumstances rendered it impossible for him to obtain them.

(*b*) **From Artificial Respiration.** If at birth a child does not voluntarily start breathing, artificial means should be adopted in order to resuscitate it. In all modern works on obstetrics two or three methods of performing artificial respiration are detailed, and for all of them are claimed cases of recovery from asphyxia neonatorum. The rationale of the proceeding is two-fold—(*a*) to introduce mechanically into the lungs a sufficiency of air to expand them; (*b*) to stimulate the dormant centre of respiration into activity, so that voluntary breathing may commence. That the latter part of the process is successful at times is proved by the very numerous cases of recovery, of which it is useless to give illustrations; the degree to which the former part is successful, or may be successful, is well illustrated by the following case:—

A full-term child was delivered still-born, and there was no effort at respiration. An attempt was made to resuscitate the child, but unsuccessfully, by blowing air into the lungs through a catheter. On inspection the lungs were observed to be of large size, but they did not present the usual appearance of lungs which had breathed. Although about three-fourths of the organs had received air by inflation, they were of a pale-fawn colour, like the thymus gland. The air was contained in the minute air-cells. They floated on water as well as all the pieces (fifteen or sixteen) into which they were divided. Sir Thomas Stevenson confirmed this observation. When compressed between the fingers under water small bubbles of air escaped; but no amount of compression short of destroying their structure caused these pieces to sink.

There is no wholly satisfactory method by which we are able to distinguish between lungs which have been partially (or even wholly) distended by artificial as opposed to natural means. Medical men must recognise this, and not attempt to stretch evidence beyond reasonable limits. In one case a child was found with its head cut off and the lungs contained air, but the mother asserted that this was due to her efforts at artificial respiration. The inconsistency of this statement as to the means whereby she inflated the lungs was clearly proved, and the examiners did not hesitate to give a decided opinion that the air found in the lungs had been derived from the act of respiration, and not from artificial inflation. This case shows that, when a theoretical objection of this kind comes to be tested practically, it ceases to present any difficulty. It may happen, however, that another person may inflate the lungs, and if the mother has been secretly delivered, she may be wrongly accused of causing the death of the child.

In all cases in which the question of artificial respiration arises there will be the evidence of the person who performed it, or at least very good material for cross-examination of some one who alleges it was done, so that pure medical evidence on the subject is of very little value.

We see then, that these changes in chest, diaphragm and lungs are capable of proving, and do collectively prove, the extent to which air has penetrated the substance of the lungs; they can and do prove

whether such air was due to respiration (natural or artificial) or to putrefaction, but they cannot prove that this penetration was the result of breathing *after the child was completely born*, nor can they prove the negative that there was no life after birth, because there was no penetration of air and because the lungs were still fœtal in colour.

Taylor thus summed up the matter very briefly, but very much to the point.

Respiration before and during Birth. It has been already stated that the pulmonary tests prove only whether a child has or has not *breathed*. Neither the hydrostatic nor any other test can positively show that the entire body of a child was completely extruded from the body of the mother when the act of breathing was performed. A certain degree of respiration may be performed (*a*) while the child is in the womb, after the rupture of the membranes—the mouth of the child being at the os uteri (*vide vagitus uterinus*, infra); (*b*) while its head is in the vagina, either during a presentation of the head or of the breech (*vide vagitus vaginalis*, infra); (*c*) while its head is protruding from the outlet; in this position respiration may be set up in a few moments and produce an appreciable inflation of the lungs. In *vagitus uterinus* or *vaginalis* the lungs receive but a very small quantity of air, and in none of these cases do the lungs show the characteristic properties of those which have fully breathed. The death of a child, which has breathed in the womb or vagina, from natural causes before its entire birth, is a possible occurrence; but its death from natural causes before birth, after it has breathed by the protrusion of its head from the outlet, is an unusual event. All that we can say is—it may take place; but the death of a child in these circumstances would be the exception to a very general rule.

It is now certain that, for criminal purposes at any rate, the law will assume—until the contrary appears from other circumstances—that the respiration of a child, established by the best of evidence, was carried on before it was entirely born, and not afterwards. For a child to be "born alive" the whole body must be brought into the world alive; it is not sufficient that the child respires in the progress of the birth. Although the medical witness in a trial for child-murder clearly establishes the fact of respiration, and therefore of life, at the time the violence was used, this evidence is not always sufficient. Unless the witness can state also that the child had breathed *after* its body was entirely in the world, the Courts may hold that, although the child had breathed, it had come into the world dead.

Evidence of Live Birth from Crying

It is quite certain that a child may breathe without crying, but it cannot cry without breathing.

There is undoubted evidence that a child may utter a cry either in the womb or in the vagina (such a cry is known as *vagitus uterinus* or *vaginalis*), such cry being distinctly audible to bystanders, and having even been heard outside the lying-in room.

Before the fœtus is able to cry air must obtain access to the uterus, and this may occur only when the membranes are ruptured and when, by manipulation or otherwise, a free passage is made to the external air. The respiratory efforts of the fœtus occur as a result of some stimulus

applied to the surface or due to the stimulus of increasing carbon dioxide tensions in its blood.

Many cases of this phenomenon have been described,[1] and it may be accepted as a fact.

Value of the Evidence from Crying. In England, crying appears to be a proof of life, and should be regarded as such, but to be regarded as a proof of live birth it is imperative that there should be a witness that the child cried after it was fully born. In the absence of such a witness, any evidence of a cry will be merely of the same nature as evidence of breathing, and will be judged by the same rules (*vide* discussion in the previous section).

Evidence of Live Birth from Muscular Movements and Rigor Mortis

The spasmodic twitching of the muscles of the mouth has been judicially regarded as a sufficient proof of live birth when observed in a child completely born; *a fortiori*, therefore, the motion of a limb will be considered sufficient legal evidence of life after birth. It is to be observed that the length of time during which these signs of life continue after a child is born is wholly immaterial: all that is required to be established is that they were positively manifested. A child which survives entire birth for a single instant acquires the same civil rights as if it had continued to live for a month or longer.

In *Fish* v. *Palmer* the obstetric experts who were summoned to give evidence differed in opinion. Two experts stated that had the child been born *dead* there could have been no muscular movement in any part of its body; therefore the child had, in their opinion, been born alive, and had manifested some evidence of life after its birth. An expert called for the defence dissented from this view. He contended from the evidence that the child had not been born alive; and, in explanation of this, drew a distinction between uterine and extra-uterine life. He attributed the tremulous movements of the lips after birth to the remains of uterine life. The jury, however, under the direction of the court, pronounced by their verdict that the child had been born living, and the plaintiff thus recovered an estate of which he had been for ten years deprived.

The difficulty in such cases is due to the fact that cellular life continues after the death of the individual. The muscles may, therefore, twitch for some time after the body is dead, and it might well be an impossible task to know with certainty whether the child at a particular moment was alive or dead. Such being the case, it would appear to be more equitable to consider that the twitching of the muscles indicated life.

In other cases the child, though alive, may be apparently dead and yet prove to be alive when artificial respiration and stimulation is applied.

It is essential that actual proof of movements having been seen must be demanded from an eyewitness before they can be accepted as reliable evidence of live birth; it is inconceivable that their past existence could be proved or even suggested by the minutest examination after death.

In *R.* v. *Pill* the body of the child was rolled in a quilt and placed in a drawer; some seventeen hours later cadaveric rigidity was found

[1] Jackson, L. M., *B.M.J.* 2: 266 (1943). Sipple, J., *B.M.J.*, 2: 653 (1912). Peiser, *Monatschr. fur Geb. und Gynæ*, August, 1913.

in the muscles. A medical man stated his belief that this was a proof of live birth, as he believed it would not have occurred had the child been still-born. We now know that such a belief is entirely erroneous ; new-born children pass into *rigor mortis* after death whether live or still-born, and therefore the presence of *rigor mortis* is of no importance in deciding whether a child has or has not been born alive.

Evidence of Live Birth from the Circulation

This will be considered as follows :—

Pulsations in cord and beating of the heart.
Changes in ductus arteriosus.
Changes in ductus venosus.
Changes in foramen ovale.

Pulsations in the Cord and Beating of the Heart. In some instances, life may be indicated by the action of the heart, when owing to some accident the lungs are not properly inflated, although efforts at respiration may be made.

Cann met with a case of breech-presentation, in which the child appeared to breathe before its head was born. As the head was large, considerable force was required in order to remove the child. Artificial respiration was resorted to for twenty minutes after its birth, and, although the beating of the heart was felt during sixteen minutes of that time, no breathing occurred. It was found that the vertebræ of the neck were dislocated, and there was great effusion of blood around the spinal cord. The lungs were of a bluish-grey colour, as in the fœtal state. They were not crepitant, and did not float on water.

A woman gave birth to a female child, as she believed, in the eighteenth week of her pregnancy. The child was believed to be dead and was placed aside. Some time afterwards convulsive movements of the body were observed. These continued for half an hour, and the action of the heart was evident to the eye from the pulsation it communicated to the chest as well as to the hand. There was no visible respiration at any time, but there could be no reasonable doubt that this child was *born alive*. In another case, the navel-string ceased to pulsate eight minutes before entire delivery. The child was born apparently dead : it was corpse-like in appearance, and its limbs were flaccid. By means of a hot bath, inflation of the lungs continued for twenty minutes, the lips acquired a slight colour, and there was a feeble sigh. After the inflation had been continued for three-quarters of an hour, the lips and face became more tinged, and respiration was established.

A case occurred in which a fœtus, born at the fifth month of uterine life, respired feebly and at intervals for about twenty-eight minutes. The child, which weighed only one pound and three-quarters, uttered no sound. The only evidence of life was in the action of the heart and the maintenance of the placental circulation. The latter gradually got weaker, and the moment it ceased, life appeared to be extinct. The entire lungs sank in water. When cut into pieces, only two small portions from the right lung floated.

Cases have been reported of children who have lived for some hours [1, 2] and yet after death the lungs were in a state of atelectasis and sank when placed in water. These cases do not indicate that breathing was not established but that it was insufficient to inflate the air vesicles. No child can survive for more than a few minutes unless air obtains access to the blood. It is quite unnecessary to prove that the lungs have been expanded in order to state that a child has been born alive. If pulsations of the cord or pulsations of the heart or movement of the chest are observed after the child has been fully extruded from the mother, that

[1] *B.M.J.*, 2 : 1567 (1900).
[2] *B.M.J.*, 1 : 146 (1901).

testimony is sufficient to prove live birth, whatever the state of the lungs may be. Auscultation of the chest should be carried out if a medical man is present, for the beating of the heart may be heard even when no external sign of cardiac activity can be observed.

In *Brock* v. *Killock*,[1] the medical man and nurse present at the delivery were eyewitnesses not wanting in experience, and they distinctly observed pulsation in the cord. The Court held that proof of breathing was not necessary, and held that there was sufficient legal evidence of life after birth in the pulsation of the cord observed by the accoucheur. This decision is in accordance with common sense. Pulsations indicate an action of the heart, as much as motion of the chest indicates an action of the intercostal muscles.

Ductus Arteriosus (Arterial Duct) and Foramen Ovale. As a result of the establishment of respiration, and the circulatory changes which are initiated thereby, it is usually found that the communication between the auricles of the heart by the foramen ovale becomes closed; and that the vascular ducts, after gradually contracting, become obliterated, and are converted into fibrous cords. There are serious objections to any conclusions as to live birth based on the state of these fœtal vessels; their closure, as a natural process, takes place slowly, and sometimes is not completed until many years after birth.

The ductus arteriosus is a vessel about half an inch long, which in the fœtus forms a direct communication between the pulmonary artery and the aorta; it conveys the larger proportion of the blood from the heart to the aorta without passing through the lungs. As soon as respiration is established, its function is at an end, and it begins to close.

Although the closure may take place as a result of the establishment of respiration, the time of its closure after birth is so uncertain as to render any evidence derivable from the non-closure altogether fallacious. The author examined the bodies of several children that had survived birth for some hours, and was not able to discover any perceptible alteration in the diameter of the duct either at its aortic or pulmonary end. In other cases partial contraction has been apparent. As the closure depends on a diversion of blood through the lungs, so it follows that, when respiration is feeble or imperfect, the duct will be found either of its natural patency, or, if closed, the closure must be regarded as an abnormal deviation. It appears that the duct may occasionally become contracted and even obliterated before birth, and before the child has actually breathed. In these cases there has been, in general, some abnormal condition of the heart or its vessels; but this, even if it existed, might be overlooked in a hasty examination: hence the contracted or closed condition of the duct cannot be taken as proof that a child has been born alive or survived its birth. In 1847 Chevers laid before the London Pathological Society the case of a child born between the seventh and eighth months, in which this vessel was almost closed, being scarcely one-twelfth of an inch in diameter, and capable of admitting only the shank of a large pin. The tissues of the duct had altogether an appearance of having undergone a natural process of contraction; and its state proved that its closure had commenced previously to birth. In fact, the child survived only *fifteen minutes*; whereas, according to ordinary

[1] 3 Giff. 58.

observations, the medical inference might have been that this child had lived a week. In this case the heart and lungs were in their normal or natural state.[1] On the other hand, the open or pervious condition of the duct is consistent with the child's having breathed after birth ; it sometimes remains pervious for many years.

The medical evidence derivable from the condition of the ductus arteriosus in a new-born child was submitted to a rigorous examination in the following case :—

The body of a child was found in a bag which had been buried in the sands on the seashore at Ayr, with such marks of violence about it as left no doubt that it must have been deliberately and intentionally destroyed. Independently of severe injuries to the throat externally, the mouth and throat internally were found to be so closely stuffed with tow and other substances that there was some difficulty in removing them. The body when found was much decomposed ; the brain was pulpy, and the cuticle as well as the bones of the skull, were easily separated. The weight of the body was seven pounds, and the child had the characteristics of maturity. The accused had been delivered of a child about three weeks before the discovery of the body, and she was charged with having caused its death. The material question was one of identity. The bag in which the body was found was part of the covering of a cushion belonging to the mother of the accused. The following appearances were met with :-The heart and lungs weighed one ounce ; the latter organs were collapsed ; the right lung was considerably decomposed, and sank when placed on water ; the left was of red colour, firm in texture, and floated on the surface when immersed in a vessel filled with water ; but on pressure there was no crepitation. The right side of the heart was filled with coagulated blood, the foramen ovale being partially open, and the ductus arteriosus impervious. The liver was large and of a leaden hue, the ductus venosus almost obliterated, and meconium was found in abundance in the lower bowel. The medical men were of opinion, from the perfect conformation of the child's body and the above-mentioned appearances, that it had been born alive. Not more than *five hours* could have elapsed from the birth of the child to the time at which its body was buried ; and that, assuming it to have been born alive, there was the strongest reason to believe it did not survive its birth more than *ten minutes*. The results of experiments on the lungs were not alone sufficient to show that the child had been born alive. The organs were light, and not crepitant ; the right lung was decomposed, and yet it sank in water, while the left was firm, and floated. A man lodging in the accused's house distinctly heard the child cry. He slept in the same room with the accused on the morning on which she was delivered. In these circumstances, the defence was that, considering the state in which the ductus arteriosus was found, this could not have been her child, because, if destroyed after being born alive, it must clearly have been destroyed immediately after birth. In that case the ductus arteriosus could not have been found impervious—*ergo*, the body found was not the body of the accused's child. It was contended that, according to all previous experience, the duct, except as a result of congenital disease, could not be found impervious in a child which had ceased to live within a *few minutes*, or even a few hours, after birth. One medical witness for the prosecution admitted that it required some days or weeks for the duct to become impervious ; but a case was reported by Beck in which it had closed within a day. Another stated that it is usually a considerable time before the duct becomes closed. Medical evidence was given for the defence, that the earliest case of closure was twenty-four hours ; and from the state of the duct in this case, the child must have survived for one day at least, or not much less. Another witness stated that the discovery of the closure in a body would lead him to infer that the child had survived three or four days. According to this evidence the body produced could not have been that of the accused's child. The jury, however, found that the child had been born alive, but that murder had not been proven.

From the evidence given at the trial the child had been born alive, it was the child of the accused, and it could have survived its birth only

[1] *Med. Gaz.*, vol. 39, p. 205.

a few minutes. The medical evidence showed that the child had been destroyed by violence. The facts that the mouth and throat were firmly packed with tow, and that there had been copious effusions of blood in the seats of violence, admitted of no other explanation. To what, then, was the early closure of the duct in this case to be referred ? There is no instance on record of the arterial duct becoming *impervious* within a period of five or six hours (in this case only as many minutes could have elapsed) after birth. Its closure is naturally the result of free and perfect breathing in a healthy child ; but the state of the lungs in this instance showed that respiration had neither been full nor complete. It is probable, therefore, that the case was similar to that described by Chevers, and that there was an abnormal condition of the duct. Either this must be assumed, or the closure must have depended on causes other than perfect respiration ; but experience shows, as a general rule, that it proceeds *pari passu* with this process.

Admitting that this abnormal state of the duct, *i.e.*, its closure previous to birth, is in general accompanied by malformation either of the heart or of the great vessels connected with it, yet Chevers' case, already related, proves that this is by no means a necessary accompaniment. Hence, the better rule will be to place no confidence on a contracted condition of this duct as evidence either of live birth or of the time during which the child has lived. It can only have any importance as evidence when the death of a child speedily follows its birth ; and these are precisely the cases in which a fallacy is likely to arise, for the contraction or closure may be really congenital, and yet pronounced normal. If a child has lived for a period of two or three days (the time at which the duct naturally becomes contracted or closed), then evidence of live birth from its condition may not be necessary ; the fact of survivorship may be sufficiently apparent from other circumstances. Hence, this species of evidence is liable to prove fallacious in the only instance in which it is required, and the Ayr case shows the dangerous uncertainty which must attend medical evidence based on the closed condition of the duct.

Ductus, or Canalis Venosus. This is the branch of the umbilical vein which goes directly to the inferior vena cava ; there is no known instance of the obliteration of this vessel previous to birth. When respiration is fully established, it collapses, and becomes slowly converted, in a variable period of time, into a ligamentous cord or band, which is quite impervious. There is no doubt that in those cases in which it is stated to have become obliterated in children that could have survived birth only a few minutes or hours, the mere collapse of the coats has been mistaken for an obliteration of the canal. It is probably not until the second or third day after birth that its closure begins ; although nothing certain is known respecting the period at which it is completed. The condition of this vessel, therefore, can throw no light upon those cases of live birth in which evidence of the fact is most urgently demanded.

Changes in the Foramen Ovale. The foramen ovale is a large oval opening placed at the lower and back part of the partition between the right and left auricles of the heart. It is considered to attain its greatest size at about the sixth month.

At an early period of fœtal life there is no valve to the foramen ovale. About the twelfth week the valve rises upon the left side of the entrance

of the vein, which thus comes to open into the right auricle. The separation of the two auricles is at the same time rendered more complete by the gradual advance of the valve over the foramen ovale, but the passage nevertheless continues open until after birth. Another valvular fold is formed on the right of the opening of the inferior vena cava, between it and the superior vena cava. This is called the Eustachian valve.

As a general rule, this valvular opening between the right and left sides of the heart exists during fœtal life, and becomes gradually closed after the establishment of respiration. It is, however, often found open in children that have survived birth several hours; and the period of its closure is as variable as in the case of the ductus arteriosus. Hence, it is not capable of supplying with certainty evidence of live birth in those instances in which this evidence is most required, and therefore the patency or closure of this aperture possesses no longer any importance.

The circumstances connected with the closure of these fœtal vessels have been statistically investigated. Facts prove that the vessels peculiar to the fœtal circulation remain open as a rule for some time after birth, and that it is not possible to determine accurately, by days, the period of their closure. The closure commenced and was often completed in the ductus venosus before it manifested itself in the other vessels. The complete closure, in by far the greatest number of cases, takes place within the first six weeks after birth, and the instances of obliteration before birth, or before the period mentioned after birth, must be regarded as rare exceptions.

The result of this **docimasia circulationis** is essentially negative: it either proves nothing, or it may lead a medical witness into a serious error.

Evidence of Live Birth from the State of the Alimentary Canal

Evidence of live birth may sometimes be derived from the discovery of certain liquids or solids in the stomach and intestines, such as milk or farinaceous or saccharine articles of food; for it is not at all probable that these substances should find their way into the stomach or intestines of a child which was born dead.

Robinson has made some researches on the contents of the fœtal stomach during uterine life. He finds that the substances which naturally exist in the stomach of a fœtus before birth are of an albuminous and mucous nature. His observations were made on the stomachs of two human fœtuses, and on those of the calf, lamb, and rabbit. The conclusions at which he arrived were:—1. That the stomach of the fœtus during the latter period of its uterine existence invariably contains a peculiar substance, differing from the uterine liquid (liquor amnii), and generally of a nutritious (?) nature. 2. That in physical and chemical properties this substance varies in different animals, being in no two species precisely similar. 3. That in each fœtal animal the contents of the stomach varies at different periods, in the earlier stages of its development consisting chiefly of liquor amnii, to which the other peculiar matters are gradually added. 4. That the liquor amnii continues to be swallowed by the fœtus up to the time of birth, and consequently after the formation of these matters, and their appearance in the stomach. 5. That the

mixture of this more solid and nutritious substance with the liquor amnii constitutes the material submitted to the process of chymification in the fœtal intestines. He considers the contents of the alimentary canal to be chiefly derived from the salivary secretion, and that gastric juice is not secreted until after respiration has been established.

Starch. In a new-born child the presence of farinaceous food in the contents of the stomach may be proved by microscopic and chemical tests, which, if positive, indicate that the child has been fed. A portion of the contents of the stomach should be placed on a glass slide, diluted with a little water if viscid, and examined under the microscope, with a power of about 300 diameters. The granules (if present) may then be distinctly seen, having the shape peculiar to each variety of starch, and not infrequently mixed with epithelial scales derived from the mucous membrane. By the addition of iodine water their shape and size will be brought out by the intensely blue colour which they acquire.

Sugar. In one case which Taylor was required to examine, the presence of sugar was readily detected in the contents of the stomach by the application of Trommer's test. In reference to the application of the sugar test, it must be remarked that starch is easily convertible into a sugar by a chemical action of saliva or mucus, so that the test may appear to indicate sugar in small quantity, when the result may be due to the presence of some converted starch.

Milk. This liquid may be found in the stomach of a new-born child, and may be identified microscopically in the fluids of the stomach by the numerous and well-defined oil globules which it contains. It is not possible nor is it necessary to distinguish human from cow's milk in these circumstances. When milk is present, milk sugar is generally found in the contents of the stomach by the sugar-test. The casein of milk is rapidly coagulated by the gastric juice, so that the casein may be found in small soft masses adhering to the lining-membrane of the stomach.

Epithelial Scales. The epithelial scales commonly found associated with articles of food in the stomach are of various shapes and sizes: they are flat, oval, or rounded, and sometimes polygonal. They are nucleated, and from their pavement-like appearance they are called "tessellated." If such are found clearly originating in the mouth, they prove an act of swallowing.

Besides the substances mentioned, other solids and fluids, such as blood and meconium (the fæcal discharges of the fœtus), may be found in the stomach of a new-born child, and a question may arise whether their presence indicates that the child was fully born. It is not impossible that a child might be fed and exert a power of swallowing when its head protruded from the outlet, and its body was still in the body of the mother. Children have been known to exert a power of sucking or aspiration in these circumstances, and with this a power of swallowing might be exercised. That the starch, sugar, or milk, etc., found in the stomach should have been given to a child when the body was only half born, is an improbable hypothesis. When the substances found in the stomach are not in the form of food, but are fluids connected with the child or the mother, the case is different. These may penetrate into the

lungs or stomach during birth, either by aspiration or the act of swallowing: they thus indicate that the child was living, but they do not necessarily show that its body was entirely in the world when they were swallowed.

Blood. An instance is related by Döring in which a spoonful of coagulated *blood* was found in the stomach of a new-born child. The inner surfaces of the gullet and windpipe were also covered with blood. Döring inferred from these facts that the child had been born alive: for the blood in his opinion could have entered the stomach only by swallowing, *after* the birth of the child and while it was probably lying with its face in a pool of blood. Taken alone, however, such an inference would not be justifiable from the facts as stated. Blood might be accidentally drawn into the throat from the discharges of the mother during the passage of the child's head through the outlet, and yet the child may not have been born alive. The power of swallowing may be exerted by a child during birth either before or after the act of breathing. This power appears to be exerted even by the fœtus *in utero*. For tests for blood *vide* Vol. I, pp. 396, *et seq.*, remembering that fœtal blood contains a proportion of nucleated red corpuscles.

Meconium. The name meconium is applied to the excrementitious matter produced and retained in the intestines during fœtal life. It is a mixture of bile-coloured granules of epithelium from the mucous membrane of the intestines, of mucous matters probably derived from a destruction of the epithelial cells, and of cholesterin crystals.

Meconium is generally discharged from the bowels of a child within forty-eight hours after birth, or at the latest on the third day. It then appears of the consistency of honey, of a very dark-green (almost black) colour, with very little yellow colouring-matter in it. It has no disagreeable odour. Its specific gravity is 1·148. It may be found in the stomach of a new-born child, and a question will thence arise whether its presence there should be taken as a proof of entire live birth. It may be discharged from the child during delivery in cases in which there is a difficulty or protracted labour. In the act of breathing it may enter the throat with other discharges, and thus be found in the stomach. That a breathing child can thus swallow meconium cannot be disputed, but, assuming that in the body of a child which has not lived to breathe, this substance is found in the air-passages and stomach, how is the conclusion affected? In the following case Fleischer was required to examine the body of a new-born child which was said to have been born dead.

He found meconium in the large intestines (the colon and rectum), and a greenish-yellow-coloured liquid in the cavity of the stomach, in the larynx, windpipe, and gullet. In the air-passages it was in well-marked quantity. The lungs contained no air, but possessed all the usual fœtal characters. When cut into pieces and placed on water, all the pieces sank. It appeared that a woman was present at the birth, who observed that the child did not breathe, but was born dead. It was not bathed or washed, and no air was blown into its lungs. From the general appearance and properties of the liquid found in the stomach and air-passages, Fleischer had no doubt that it was meconium from the intestines of the child. It could not have been swallowed after the child was born, but must have been accidentally drawn into its throat by efforts to breathe during birth. Some of the meconium had probably been discharged from the bowels of the child during labour, and as the mouth passed over this liquid a portion was drawn into the throat by aspiration. When once there, the instinctive act of swallowing would immediately convey a portion of it into the stomach. The same remark applies to the urine.

As the facts connected with the birth were well known, this appears to be the only reasonable explanation.

Meconium may be generally recognised by its dirty-green colour and general appearance, as well as by the absence of any offensive odour, which it does not acquire until after the third or fourth day from birth, when it becomes mixed with feculent matter. In the air-passages it is sometimes associated with vernix caseosa, and hairs derived from the skin.

But little need be said of its chemical properties ; still, as the detection of stains of meconium on clothing may occasionally form a part of the medical evidence, a few observations are here required. The stains which it produces are of a brownish-green colour, very difficult to remove by washing. They stiffen the fabric, and are usually slightly raised above the surface, without always penetrating it. Meconium forms with water a greenish-coloured liquid having an acid reaction, and a boiling heat does not affect the solution. Nitric acid, and also sulphuric acid and sugar, yield with it the green and red-coloured compounds which they produce with bile. Cholesterin may be separated from it by hot ether.

It may be remarked, in reference to stains produced by the fæces of a child which has survived birth, that until the fifth or sixth day they retain a dark-green or greenish-yellow colour. On the seventh day after birth, they generally acquire a bright yellow colour, like that of the yolk of egg ; and this colour, if the child is in health, they will retain during all the time that it is suckled.

The presence of stains of meconium on the clothing of a child was once considered, in the absence of any evidence from the lungs, to furnish sufficient proof that a child has been *born alive*.

The body of a child, completely dried or mummified, was found concealed in a space in the chimney of a house. From the dry state of the body it had apparently been there for a considerable time. It had the features of a mature female child. It was wrapped in linen, which was marked by two kinds of stains, some of a deep-green almost black (meconium) and others of a reddish-brown colour (blood). The internal organs had been completely destroyed, chiefly by larvæ of insects of which many of the dried chrysalis cases were found. The skin was dried to a parchment condition. Was this child born alive ? As the lungs were destroyed attention was directed to the meconium stains on the linen ; and it was concluded from these that, had the child died before or during labour, the greater part of the meconium would have been discharged before birth. Assuming that a quantity of it still remained in the bowels, this could not have been discharged from them, as a result of vital contractility after death. Further, the portion of linen around the nates of the child was not stained, hence there had been no discharge *post-mortem*, after the dead body had been placed in the chimney—leading therefore to the conclusion that the linen had been stained by the natural discharge from a child born living, and previous to the disposal of its body. It was inferred, from the large quantity of meconium, that it had been discharged during a state of severe suffering resulting from a violent death. The opinion was given : 1. That this mummy-child was mature ; 2. That it was born alive, and that it died from violence soon after its birth ; and 3. That its death probably took place about two years before the discovery of the body. The latter conclusion was based on entomology, *i.e.*, on the condition of the chrysalis cases and the larvæ of the *Musca carnaria* found in the cavities of the body. The facts were such that a shorter period than two years would not account for the state in which the insects were discovered. Upon this evidence a woman was tried before the Jura Court of Assizes, on a charge of child murder but was acquitted.

This case is left in the text as it is sometimes quoted, but there was no evidence of live birth, for the stains of meconium on the linen might be accounted for irrespective of this theory. There was no evidence of murder, for all the facts admitted of an explanation on the assumption that the child had been either still-born or, if born living, that it had died from natural causes soon after its birth, and that its body had been concealed in the spot where it was found.

Foreign Substances in the Air-passages and Stomach. Maschka met with the following case :—

A woman was secretly delivered of a child, which she alleged was born dead, but she did not produce its body until after the lapse of fourteen days, when it was found in such a state of putrefaction that no satisfactory evidence of live birth was obtained from the lungs. These organs, as well as the heart and liver, contained small bladders of air from putrefaction and floated on water. On slight compression, the lungs sank. The air-passages, gullet, and stomach contained sand and excrementitious matter, which was pressed out of them on a section being made. The air-passages were so blocked up as to furnish a sufficient cause for the prevention of breathing and for death from suffocation. The woman, when charged with the murder of her child, confessed that she was suddenly delivered while having, as she supposed, an evacuation—that she fainted, and that when she recovered, she found she had been delivered of a child, which had fallen into the privy and was dead. The medical evidence was in accordance with this condition of the body. Maschka concluded that the child had come living into the world, and had died from suffocation. He drew this inference from the discovery of excrement and sand in the air-tubes, lungs, and stomach. He considered from the appearances, that in the aspiratory effort to breathe (a living action) the child had drawn these substances into the lungs, and, further, that they could have found their way into the stomach only by the act of swallowing. These actions could not have taken place until after birth, and in his judgment they clearly proved that the child had come living from the body of the mother.

In one case a woman was suddenly delivered of a child while sitting over a slop-pail of dirty water. On examining the body, it was obvious that it had not breathed. There was no air in the lungs, but a quantity of dirty water like that in the pail was found in the stomach. This could have entered the organ only by the act of swallowing, and, in Ramsbotham's opinion, the child had swallowed the liquid under some fœtal attempts to breathe. The coroner who held the inquest directed the jury that the child was born dead ; but most physiologists will consider that the power of swallowing cannot be exerted by a dead child ; and as its body must have been entirely delivered in order to have fallen into the liquid, there was proof that it had been born living, and that in this instance it had died after it was entirely born, by the prevention of the act of breathing.

The inference of live birth in these cases was based on good physiological grounds. The discovery of foreign substances, which from their nature could not have entered the body during delivery, is a good proof of live birth, but we ought to be well assured that such substances could not have accidentally found their way into the body after death. Thus it might be suggested in defence that they had penetrated into the stomach and lungs as a result of putrefaction, if the body was immersed in liquid. It will be for the examiner to determine, by a proper examination at the time, how far this can explain the facts. The discovery of excrementitious matter in the interior of the stomach and in the substance of the lungs was a proof that the child had exerted the acts of swallowing and aspiration.

If the body of a child is so putrefied as to lay open the stomach and lungs so that foreign matters can have free access to them, it would of course be impossible to base an opinion on these conditions.

The condition of the **urinary bladder,** whether empty or full, is of no medico-legal value ; it may be emptied before birth or at birth.

Value of Evidence from the Alimentary Canal. The discovery of farinaceous food, milk, or sugar in the stomach will furnish almost conclusive evidence of live birth, since substances of this kind are not found naturally in this organ, and it is almost inconceivable that anyone should try to feed a dead baby or force food down its throat without leaving evidence of such an attempt.

The presence of natural fluids—such as *blood*, *meconium*, or the watery discharge attending delivery—in the stomach and air-passages of a new-born child, does not prove live birth, but merely indicates the existence of some living actions in the child at or about the time of its birth. It is otherwise with foreign substances, which according to their source will suggest the reason for their presence.

Evidence of Live Birth, etc., from the Umbilical Cord

The points which should be noticed about the cord in all cases are :—

Pulsations or their absence during or after the birth.
Mummification of the cord.
The line of separation at the navel.
The manner in which it has been severed.
Whether and by what it has been tied.
Its total length if available.

Pulsations in the Cord. Since these are caused by the pulsations of the fœtal heart, they are certain evidence of live birth, and their complete and continuous absence at birth indicates dead birth. Such pulsations can be sworn to only by one who was in attendance at the birth, for neither their presence nor their absence for a few minutes would leave the slightest trace that could be discovered even by the most painstaking autopsy.

Mummification of the Cord. In a child that has been born alive, or has survived its birth for a period of from twelve to twenty-four hours, that portion of the umbilical cord which is attached to the abdomen undergoes certain changes ; thus it dries and becomes slowly shrivelled, and in from three to five days it separates from the body with cicatrisation of the wound of separation.

The cord does not separate at the part which is tied, but close to the abdomen. It separates generally within five days, by a process of sloughing ; the skin connected with the dead portion of cord presenting a red line arising from capillary congestion. During the separation the umbilical vessels are gradually closed. The obliteration of these vessels is effected in a peculiar manner. The calibre diminishes as a result of a concentric thickening of the coats, so that, while the vessel retains its apparent size, its cavity is gradually blocked up. A quill would represent the form of the vessel in the fœtal state, and a stem of a tobacco-pipe in the obliterated state. It is only by cutting through the vessel that the degree of obliteration can be determined.

The state of the *umbilical cord* has often furnished good evidence of live birth, when the other circumstances of the case were inadequate to

furnish decisive proof. In the following instance it might have been suspected, but for the state of the cord, that the child had been still-born, and that its lungs had been artificially inflated.

In consequence of some suspicion respecting the cause of death, the body of a child had been exhumed soon after burial. It weighed nearly five pounds, and was eighteen inches long; the opening for the navel was exactly in the centre of the body. The hair on the scalp was about an inch in length, and plentiful; the nails reached to the extremities of the fingers and toes. There was no mark of violence about it. The *navel-string had separated* by the natural process, but the skin around it was not quite healed. The tendon of one of the muscles of the leg was prominent, and apparently contracted at the instep. The left testicle alone had descended into the scrotum—the right was still in the inguinal canal. This rendered it probable that the child had not quite reached maturity. It was by the peculiarity of the instep that the body of the child was identified. In the first instance the body of another child had been brought from the same burial-ground, but rejected, from the absence of this appearance of the foot (*cf.* "Identity," Vol. I). On opening the chest, the lungs were observed to be situated at the back part and not filling the cavity. They weighed together 861 grains—the right weighing 430, and the left 431 grains. The heart, thymus gland, and lungs were placed together in water, but they immediately sank. The lungs, when separated from the other organs, floated, but with a slight degree of buoyancy. Indeed, this was established by the fact that they sank with the heart and thymus attached. The lungs were cut into twenty-two pieces: three pieces from the apex sank; the remaining nineteen pieces floated, and they were not made to sink by pressure. The foramen ovale was contracted, as well as the ductus arteriosus, to about one-half of the fœtal diameter. The bladder was perfectly empty—the intestines contained only mucus.

The conclusions at the inquest were:—1. That the child had been born alive, and had lived certainly not less than three days, and probably longer. 2. That respiration during that time had been but imperfectly established. 3. That in all probability the child had died a natural death. The conclusions were well warranted by the facts. Experiments on the lungs were not necessary, owing to the state of the umbilical cord. It was subsequently proved that the child had lived eight days after birth.

It is commonly stated that once such drying of the cord has appeared it cannot be removed by soaking in water, but this is not correct. Prolonged soaking causes the cord to swell up, but the plump appearance of the newly born cord is not observed.

It is stated that if a dead-born child be thrown into water the cord undergoes a liquefactive form of decomposition instead of mummification; this is true, for the drying is a purely physical change, which cannot go on when the cord is submerged. It follows that if a child be removed from the water with a mummified cord attached to it, the evidence is complete that the child was kept in a dry place for a sufficient length of time for mummification to have taken place, though it must not be assumed that a dry cord is any proof that the child lived while the cord was drying.

The Line of Separation at the Umbilicus. It is stated that even at birth a reddish ring may be noted at the insertion of the cord in the skin at the umbilicus; this disappears after death, and must not be confounded with the line of inflammation, which appears at the point in the skin from which the cord will fall. The mummified cord, being dead tissue, is separated from the living skin of the infant by a process of inflammation; this inflammation is as indubitable a sign of life as

it is possible to get: when, therefore, this ring of inflammation is found on a dead child, we have definite proof of life for at least thirty-six hours. The cord actually falls off owing to the changes in the tissues at the umbilicus at some period between the second and the tenth days, the fourth, fifth, and sixth days accounting for a very large majority of the cases; at its fall it leaves a small open wound which heals in two or three days' time. If this wound or a healed scar is found, we have further conclusive proof of life for a period to be measured by days, say from four to about twelve.

The Manner of Severance of the Cord. This in itself is not a matter of very great importance, as a rule, either to the child or to the mother, but in a medico-legal inquiry on a dead child it may suddenly assume the very highest importance as a piece of corroborative evidence; it must, therefore, be most carefully noted and recorded.

It is difficult to sever the cord by simple tearing, and if it is grasped in the hands it is so slippery that it can scarcely be twisted, unless a piece of cloth is used with which to hold it. This manipulation forces the jelly, of which the bulk of its structure is formed, into irregular heaps, so to speak, within the coverings of the cord; it might be important to notice this as corroborative of a woman's statement that she had thus severed the cord.

If the cord is cleanly cut this would tend to set aside the explanation of the child having accidentally dropped from the female, because in such an accident the cord should be found *ruptured*. The practitioner should make a careful examination of the divided ends of the cord by the aid of a lens, or a rupture may be mistaken for a section with a sharp instrument. For this purpose it should be spread out on a board or floated in water. In one case the child fell from the mother, and the cord broke spontaneously. "The torn ends were nearly as sharp-edged and flat as if cut."

This case proves that a careless or hasty examination of the ends of the cord may lead to a serious mistake.

If the cord is ruptured by natural accident, the free ends are usually irregularly lacerated, and the rupture takes place either near to the placental or the navel end, more commonly within a few inches of the navel. In twenty-one of the cases observed by Klein, it was found to have been forcibly torn out of the abdomen; but it may be torn or lacerated at any part of its length, although the rupture is commonly observed near to one extremity. Among the cases of sudden delivery which occurred to Olshausen, the cord was torn through at three inches from the navel in one instance, and no bleeding followed. In two the cord was torn through its middle, and at first there was great bleeding; in three other cases it was torn close to the navel, and no bleeding had occurred. In four instances the cord was torn at five or six inches from the navel, and there was no bleeding, although it remained untied for ten minutes.

A medical witness was once asked by the judge whether a rupture of the cord might not lead to fatal bleeding. The above facts show that a rupture of the cord is not necessarily fatal, even when the circumstances are unfavourable to the child by reason of the closeness of the rupture to the abdominal end.

Sometimes the mark of a previous cut may be found on the cord near one of its divided ends -the first cut with the scissors not having effectually divided it.

In one case where the body of a child was found in a privy, the cord had been ineffectually cut in one spot previously to its complete division in another part. The cord had also been pulled out after this cut, so as to elongate the vessels: hence they projected from one part of the sheath at one cut portion, while they were retracted in the other. This accurate observation not only showed that the cord had not been ruptured by the child accidentally falling from the mother, but it served to establish the identity of the placenta, which was found concealed at a distance from the body.

The body of a child, which was found in the soil of a privy, was putrefied, but the lungs had not undergone putrefaction. Both feet were wanting and the bones of the legs were exposed, owing to the removal of the soft parts. There were no marks of murderous violence on the head, neck or upper part of the body. About six inches of the navel-string were attached to the abdomen, and this had not been lacerated, but sharply cut through. This observation was of importance, for it suggested that the woman had not been accidentally delivered while sitting in the privy, or the cord would have been found lacerated.

The main questions were:—Did this child come into the world living, and was its death attributable to violent or accidental causes? Grains of sand and particles of coal were found upon the tongue, and in the fauces, larynx, windpipe and its ramifications, as well as in the pharynx and gullet, the mucous membrane in these parts being of a brownish-red colour. The lungs were placed at the back of the chest, the sharp edges reclining on the sides of the pericardium; they had a bluish-brown colour behind, but they were of a light red with stellated patches of redness in front. The substance of the organs was not putrefied, it was elastic when pressed and crepitated on being cut. They contained a moderate amount of blood. Both lungs floated on water entire and divided. The stomach contained an offensive dark-coloured fluid, mixed with grains of sand, pieces of coal, and other foreign matters. The conclusions drawn were that this child was mature, that it had been born alive, the navel-string designedly cut, and its body afterwards thrown into the place where it was found, for the purpose of concealment. The inference of the child having been born alive was based on the state of the lungs and the foreign substances found in the air-passages. There had also been the power of swallowing, the same substances having been found in the stomach. For however short a time, these conditions proved that the child had lived, and had breathed after it was born. The cause of death was assigned to suffocation and the prevention of breathing.

A girl, who stated that she was not aware of her pregnancy, was suddenly delivered while sitting on a night-stool. According to her account, she fainted, and on coming to herself she found the child on the floor dead. The child had fully breathed, the umbilical cord had been cut, and there was no mark of violence on the body. The cause of death was assigned to exposure, and the absence of those attentions required by a new-born child, as well as to congenital debility. The girl was found guilty of causing the death of her child by imprudence, inattention and negligence. The cutting of the cord suggested that her tale was untrue.

The Length of the Cord. Should this be available it should be recorded, for it may be of material importance if it be alleged that the child was strangled by it.

The length varies from some six or eight inches up to as much as five feet, but average figures should not be regarded when the facts of a case are ascertainable.

Evidence of Live Birth from the Appearance of the Skin

The skin of a new-born child is of a bright red colour, and is covered with a greasy material known as vernix caseosa. This observation is in itself of no importance, except that the vernix caseosa is readily removed by washing, and its presence or absence may be a point corroborating or negativing a statement that the child had or had not been washed.

Evidence of Live Birth from the Mode of Birth

It has been suggested that when a child is born by the feet, and there are full marks of respiration in the lungs, the mode of birth will at once establish that the body must have been entirely in the world in order that the breathing should have taken place. Herapath met with an instance of this kind. It is assumed that the head in these circumstances is born instantaneously, and that the child cannot breathe until the head is released from the outlet. Before such a conclusion can be drawn there should be clear evidence that the child was actually born by the feet such as the absence of a birth tumour on the head and the different shape of the head which does not have the elongated appearance found in head presentations.

If a child be born by any presentation other than a head presentation, the chances of being born dead are very materially increased.

It is possible that a woman might confess or allege that a child was born feet or buttocks first (or there might, in a civil case, be the evidence of the accoucheur). In such a case as this, it would have to be admitted that the child had been born alive if every piece of both lungs floated in performing the hydrostatic test. For *vagitus vaginalis* would not produce this result, and if once the mouth is outside when the head comes last, the rest of the head follows at once without delay.

CHAPTER X

INFANTICIDE

Infanticide Act, 1938. The unsatisfactory state of the English law of infanticide referred to in some former editions was dealt with by the Infanticide Act, 1922. That statute has been repealed and re-enacted, with amendments, by the Infanticide Act, 1938, which provides that where a woman, by any wilful act or omission, causes the death of her child, being a child under the age of twelve months, but at the time of such act or omission the balance of her mind was disturbed by reason of her not having fully recovered from the effect of giving birth to the child, or by reason of the effect of lactation consequent upon the birth of the child, then, notwithstanding that the circumstances were such that but for the said Act, the offence would have amounted to murder, she shall be guilty of the felony of infanticide, and shall be punishable as for manslaughter. If, in such circumstances, the mother of the child is tried for murder, the jury may return a verdict of manslaughter, or of "guilty but insane," or of concealment of birth, pursuant to s. 60 of the Offences against the Person Act, 1861, except that for the purpose of the proviso to that section a child shall be deemed to have recently been born if it had been born within twelve months before its death.

Legal Presumption and Burden of Proof. It is well known that many children come into the world dead, and that others die from various causes either during or soon after birth. In the latter, the signs of their having lived are frequently indefinite. Hence, in order to provide against the possibility of wrong convictions, **the law presumes that every new-born child found dead was born dead,** until the contrary is proved. The onus of proof that a *living* child has been killed is thus thrown on the prosecution; and no evidence imputing homicide can be received, unless it is first made certain, by medical or other facts, that the child survived its birth, and was *legally* a living child when the alleged violence was perpetrated upon it. Hence on these occasions there is a most difficult duty cast upon a medical witness. In the greater number of cases the woman is delivered in secrecy, and no one is present to give evidence respecting the birth of the child. It is in these circumstances that medical evidence is particularly important. A medical practitioner should be specially cautious in putting questions to a woman charged with infanticide.

Body of the Child not Discovered. In cases of infanticide, medical evidence is founded commonly on an examination of the body of the child; but it must be borne in mind that a woman may be found guilty of the crime, although the entire body of the child is not discovered;

for it may have been destroyed by burning, or disposed of otherwise ; and a medical witness may have only a few calcined bones to examine. In these cases of the non-production of the body, adequate legal evidence of the homicide would, however, be required ; and this evidence must be such as would fully establish a matter of fact before a jury.

The subject of infanticide[1] has received much attention from medical jurists by reason of the facility with which the crime may be committed. The reports of inquests show that many infants die in circumstances of great suspicion. One of the strongest motives for destroying the infant appears to be the desire to avoid the disgrace of having an illegitimate child. As a rule the crime is attempted only where pregnancy has been concealed, and where delivery is effected in secret. If the child has been killed secretly, the earliest opportunity is taken to dispose of the body. When the dead body of the child is hidden on the premises, discovery generally takes place. In several instances the mothers of newly born dead children have been brought before the coroner's court. There has been considerable reluctance on the part of a coroner's jury to return a verdict of wilful murder.

Infant Life Preservation Act, 1929. By this Act any person who, with intent to destroy the life of a child capable of being born alive, by any wilful act causes a child to die before it has an existence independent of its mother is guilty of the felony of child destruction, provided that it is proved that the act which caused the death of the child was not done in good faith for the purpose only of preserving the life of the mother.

Evidence in Infanticide. In giving evidence at a coroner's inquest, as much care should be taken by a medical practitioner as if he were giving it before a judge at the assizes. In practice, when the mother of the deceased child gives evidence every possible allowance is made in view of the state of her mind. A medical witness must learn to practise making accurate observations of conditions of minor importance, such as whether the body is washed or not, the method of severing the cord, the presence of scratches, nail marks, tiny punctured wounds, etc., etc. Such details may easily escape observation, but unless they are noticed and reduced to writing in the notes of the medical witness, it would be well for him to keep silent about them.

Notification of Births. By the Public Health Act, 1936, s. 203, the medical practitioner or midwife who is present at a confinement is required to notify the local medical officer of health of the fact that a living or still-born child has been delivered of a particular woman. By the Births and Deaths Registration Act, 1926, the birth of every still-born child must be registered by the registrar in a register of still-births containing the heads of information prescribed in the First Schedule to the Act.

The Midwives and Maternity Homes Acts, 1902 to 1926, make provision for the keeping by the Central Midwives Board of a roll of midwives and for the authorising of the use of midwives' badges. These Acts and the Nursing Homes Registration Act, 1927, and the Public Health Act, 1938, provide for the registration with and inspection by the local supervisory authority of all maternity and nursing homes. "Christian Science" nursing homes may be exempted from these provisions (Public

[1] See paper by J. C. M. Matheson in the *Medico Legal Review*, 1941, p. 135.

Health Act, 1936, s. 193). Any person who keeps a maternity or nursing home without licence or registration is liable to be fined 50*l*., or, in the case of a second or subsequent offence, to imprisonment up to three months.

Adoption of Children. Until the passing of the Adoption of Children Act, 1926, the law of England did not recognise the adoption of children. It is now possible, on conditions laid down in the Act, to obtain an adoption order of the court for the adoption by the applicant of a child under the age of twenty-one, provided such child has not been married.

NATURAL CAUSES OF DEATH AT OR BEFORE BIRTH

These will be considered in the following order :—

Statistics of children born dead.
Absence of skilled assistance.
Immaturity or debility.
Malformations incompatible with life.
Spasm of the larynx.
Diseases acquired *in utero*.
Diseases of the placenta.

Proportion of Children Born Dead

On any individual case of infanticide this can have but little influence as evidence ; the figures themselves are, however, of considerable interest.

It is well known that of children born in ordinary circumstances, a great number die from natural causes either before or during birth, or soon after.

Like the infant mortality rate as a whole, the still-birth rate and the neo-natal mortality rate vary considerably from country to country, but they are always highest among the less fortunate sections of the community. The likelihood of still-birth or neo-natal death is greater in a first than in a subsequent pregnancy, and in cases where the mother is unattended and unaided at her confinement. These facts should be borne in mind when we are estimating the probability of the cause of death being natural. In most cases of alleged child murder the woman is primiparous and the child is illegitimate.

The chief causes of death in still-born infants and of neo-natal death are indicated in the following tables, which are based on the careful study of more than a thousand cases by a pathologist with exceptional experience in this type of investigation[1]:—

TABLE 1

CAUSES OF FOETAL DEATH IN 435 CASES OF STILL BIRTH

Condition	Full Time	Premature	Total	Per cent
Asphyxia	94	68	162	37·2
Intracranial Hæmorrhage ..	68	37	105	24·1
Developmental Defects ..	23	65	88	20·2
Infections	11	3	14	3·2
Miscellaneous	10	11	21	4·8
Not ascertained	12	33	45	10·3

[1] Macgregor, Agnes R., Brit. Med. Bull., Vol. 4, No. 3, 1946.

TABLE 2
CAUSES OF 454 NEO-NATAL DEATHS (WITHIN THE FIRST WEEK AFTER BIRTH

Condition	Full Time	Premature	Total	Per cent
Intracranial Hæmorrhage ..	29	133	162	35·7
Asphyxia	14	67	81	17·8
Infection	20	42	62	13·5
Developmental Defects ..	37	21	58	12·8
Prematurity only	—	50	50	11·0
Miscellaneous	16	22	38	8·6
Not ascertained..	3	—	3	6·6

An important feature of this investigation was the demonstration of the important part which infection plays as a cause of neo-natal death. Although only 3·2 per cent. of still births could be attributed to infection, the figure rises to 13·5 per cent. for deaths occurring during the first week of life, and to the very high proportion of 78 per cent. for those deaths occurring between the end of the first week and the end of the first month of life. In the great majority of cases, the infection is of the respiratory system, and, particularly in those which prove fatal within the first few days, there is commonly associated a history of difficulty at birth, with the possibility of partial asphyxia and the inhalation of amniotic fluid. In many of these cases the death is sudden, with no preceding clinical picture of pneumonia such as might be expected in the older child or in the adult, and this must be borne in mind, not only with reference to suspected infanticide cases, but in all cases involving the sudden, unexpected death of young infants.

The figures given above also demonstrate the great importance of prematurity as a factor in causing neo-natal death.

The underlying condition responsible for intrauterine death of the fœtus are indicated in the following table of the causes of still birth in Scotland for the year 1939.[1] (No figures are published for England and Wales.)

Hazards of Birth	No.	Per cent.	Pre-existing at Birth	No.	Per cent.
Difficult labour :			Fœtal deformity	529	13·8
Torsion of cord ..	95	2·5	Antepartum hæmorrhage :		
Prolapse of cord ..	185	4·8	Placenta prævia ..	150	3·9
Malpresentation ..	285	7·4	Accidental hæmorrhage	258	6·7
Pelvic deformity ..	64	1·7	Antepartum hæmorrhage (not defined) ..	87	2·3
Prolonged labour and uterine inertia ..	188	4·9	Toxæmia	342	8·9
Injury at birth ..	72	1·9	Chronic disease of mother	83	2·2
Other specified causes ..	236	6·2	Ill defined :		
Ill-defined :			Debility	69	1·8
Asphyxia	341	8·9	Atelectasis	24	0·6
			Macerated fœtus ..	175	4·6
Total ..	1,466	38·3	Prematurity	286	7·5
			Unknown	363	9·5
			Total ..	2,366	61·7

[1] Infant Mortality in Scotland : Dept. of Health for Scotland : H.M. Stationery Office, 1947.

In every case of child murder and of infanticide death is presumed to have taken place from some such cause as is indicated above, unless and until the contrary appears from the evidence. This throws the onus of proof entirely on the prosecution. Many children die before performing the act of respiration ; and thus a large number come into the world dead or still-born. The proportion of *still-born* among legitimate children, as it is derived from statistical tables extending over a series of years, and embracing no fewer than eight millions of births, varies from one in eighteen to one in twenty of all births.[1]

Should breathing be established by the protrusion of the child's head from the outlet during the birth of the body, the possibility of death from natural causes is considerably diminished. Nevertheless, a child may breathe and die. Many years ago Hunter wrote : " We frequently see children born who, from circumstances in their constitution or in the nature of the labour, are but barely alive, and after breathing a minute or two, or an hour or two, die in spite of all our attention. And why may not this misfortune happen to a woman who is brought to bed by herself ? " In the present day a charge of child murder or of infanticide is rarely brought, except where there are the obvious marks of severe and mortal injuries on the body of the child.

Absence of Skilled Assistance

In such criminal cases as are here being considered, *i.e.*, those in which there can arise or has arisen a doubt whether the child was born alive, the absence of skilled assistance is a common occurrence. Skilled assistance might and probably would prevent the following conditions from occurring :—

(*a*) **Prolonged Labour.** For the actual causes of this a text-book of obstetrics should be consulted ; a child that is slightly feeble and delicate is very likely to die from this cause. If this be alleged, its occurrence may be corroborated if a sanguinolent or serous tumour (called *cephalhæmatoma*, or *caput succedaneum*, *vide* p. 195) is found on the head of a child, and the head itself is deformed or elongated ; internally, if the vessel of the brain be in a congested state the existence of deformity in the pelvis of the woman might corroborate this view ; but in primiparous women (among whom charges of child murder chiefly lie) with well-formed pelves, delivery is frequently protracted. It is presumed that there are no marks of violence on the body of the child, excepting those which may have reasonably arisen from accident in attempts at self-delivery.

In the following case a large child obstructed labour.

A fœtus had the following measurements : total length, 23¾ inches ; circumference of neck, 10 inches ; circumference round shoulders, 20 inches ; circumference round chest, 16½ inches and circumference round pelvis at crest, 15½ inches. The fœtus weighed 30 pounds 4 ounces. The patient, a cowman's wife, three years ago gave birth to a child weighing 18 pounds. The labour was difficult but natural and not unusually long, no instruments were used, no chloroform was given and there was no injury to maternal parts. The child died during delivery. The mother believed herself to have been pregnant exactly eleven months.[2]

[1] *Brit. and For. Med. Rev.*, No. 7. p. 235.
[2] *Lancet*. May 25th. 1904.

(*b*) **Hasty Parturition.** In cases such as that in which a woman, under the impression that she was about to have a motion, sat over a large water-jug and was delivered of a child, it is proper to make allowance for a mistake which may be compatible with innocence. A woman is often unable to distinguish the sense of fulness, produced by the descent of a child, from the feeling which leads her to suppose that she is about to have an evacuation ; and thus it is dangerous, when a labour has advanced, to allow her to yield to this feeling, for the child may be suddenly born. Two cases of this description are reported, where there could not be the slightest suspicion of criminality.

In one case (a primipara) the child was born in these circumstances, but its life was saved ; if there had been no convenience other than a privy, the child would have died. In the second case (although one of third pregnancy) the woman was completely deceived by her sensations.

This alleged mistaken sensation forms a frequent and specious defence on charges of child murder or of infanticide. A medical witness knows that an accident which occurs to women in circumstances without suspicion, may also occur in suspicious circumstances without necessarily implying guilt.

A new-born child recovered after it had remained four hours in a drain-pipe connected with a cesspool which received the soil of privies. A girl was charged with attempted child murder. She had been recently delivered. She stated that she had been to the privy for a natural purpose, and was there suddenly delivered. A full-term infant was found in the large drain-pipe between the privy and the cesspool. It was alive, and was restored by a warm bath and other means. There was no mark of violence ; the cord had been ruptured as by a fall ; and there was nothing to show an attempt at murder. The appearances presented by the body of the child were consistent with the girl's statements. The preservation of its life was remarkable. The first part of the drain-pipe was wide enough to admit the body, which lodged at the lower part, near a bend. It was thus saved from falling into the cesspool. The drain-pipe contained air and no sewer gases—hence the child could breathe, and before removal it was heard to cry.

In a case reported by Wharrie, the child fell from the mother while she was sitting over a large jug containing water, and from the state of the lungs it was evident that there had been no respiration. The cord was found tied. As the child was removed from the vessel dead, the ligature must have been applied after death, and the body replaced in the jug.

Drowning may be the result of accident from sudden delivery. A woman in an advanced state of pregnancy, while sitting on a chamber-vessel, was suddenly delivered. The child fell into the fluids in the vessel, and before assistance could be rendered it was dead. A woman who had already had two illegitimate children, delivered herself of a third, and alleged that it was still-born. The body of the child was of average size. The head and face were much congested, and there was slight oozing of bloody fluid from the nostrils. The eyelids were discoloured ; the lips were separated, swollen, and livid ; the chest was arched. The navel-string had been cut but not tied, and there was a slight oozing of blood from it. The lungs had all the usual fœtal characters ; they sank in water when cut into small pieces. There was dark fluid in the heart and large vessels. The woman was delivered while sitting on the chamber utensil, when the whole contents of the womb at once passed from her—the child and after-birth with the waters. A neighbour came in and placed the woman in bed, but omitted to look to the child, which was soon afterwards found dead. A medical witness stated that the child had not breathed. It had probably been born alive, but had died from prevention of breathing at its birth, owing to the lack of proper attention.

There is no doubt that many children are thus born alive, but they do not continue to live after birth, owing to the accidental or criminal prevention of respiration. Such cases are always open to the suggestion

that they arose from accident ; and a woman charged with child murder or infanticide is entitled to have the full benefit of any doubt. Two instructive cases are reported by Carson, which show that, alone and unassisted, the mother of an illegitimate child may be placed in circumstances of the greatest suspicion, although innocent of any attempt to destroy the life of her child.

An inquest was held on the body of a newly born child which had been born during an effort to *defœcate*. The cause of death was suffocation by fæces and urine, and yet the hydrostatic test showed that the lungs were completely inflated. The woman was forty-two, a multipara ; her youngest child was eight years old.

In this connexion the question arises **whether a woman can be delivered without being conscious of it.** The signs of delivery may be discovered by a practitioner ; the offspring may also be found. She may admit the fact of her delivery, but allege that she was totally unconscious of it. This plea is occasionally raised in infanticide cases ; and as the possibility of the occurrence may be questioned, the practitioner must be provided with a knowledge of those facts which medico-legal writers have accumulated respecting it. There is no doubt that a woman may be delivered unconsciously during profound sleep, or while labouring under coma, apoplexy, asphyxia, or syncope ; or if suffering from the effects of narcotic poisons, anæsthetics, or intoxicating liquors. It is said also, that delivery has taken place spontaneously while a woman was in the act of dying. This, however, has no bearing on the present question. It is in those cases where a woman, after her recovery, pleads unconsciousness of delivery, that medical practitioners are chiefly consulted. Besides the causes enumerated, hysteria, when accompanied with loss of sense and motion, has been mentioned as a state in which parturition is liable to occur unconsciously. There need be no surprise if delivery takes place in such circumstances, inasmuch as the contractile power of the uterus is altogether independent of volition ; but, unless the morbid states already mentioned are accompanied by the most profound lethargy and entire loss of sensation, it can rarely happen that the contractions of the uterus, in its efforts to expel the child, should not at once rouse a woman into consciousness. We ought particularly to expect this in primiparæ. Parturition in some women, however, especially when the pelvis is wide and the child small, may take place with rapidity and ease and almost without pain.

When a woman has frequently borne children, delivery sometimes takes place without effort, and without any consciousness on her part. On other occasions a woman may lie in a state of torpor or stupor, or suffer from **eclamptic convulsions,** and have no recollection of her delivery. A woman may be delivered during eclamptic convulsions, which might have attacked her before labour set in ; and after delivery, but before complete recovery, she might become maniacal, during which interval she might have killed or injured her child ; or the child might have been borne dead, or an accidental injury might have occurred to it. She would with truth assert her entire ignorance of it.

A mother of nine children when attended in her tenth labour was lying calmly and placidly in bed, and was perfectly insensible. The child had been expelled with the placenta. She did not recover her sensibility for ten or twelve hours when she had no recollection of the birth, or of any circumstances connected therewith ; she suffered no pain or uneasiness.

Profound lethargy occasionally makes its appearance about the time of delivery.

A woman remained in a state of sleep for three days, and was delivered while in this unconscious condition: on awakening, she had no recollection of having suffered any pain during delivery. The mother of several children, on one occasion, was unconsciously delivered during sleep. In another case labour commenced and progressed in a woman to the second stage during sleep.

The results obtained by the use of anæsthetics show that the expulsive efforts of the uterus are as energetic in the unconscious as in the conscious state. It may appear extraordinary, however, that a primiparous woman, unless rendered unconscious by narcotic substances, should be delivered without suffering pain; nevertheless, a case of this kind is recorded.

The woman's age was twenty-one; she had been in labour about six hours; she complained of no pain, and the child was born without effort or consciousness. The child was healthy but small, weighing rather more than four pounds. A healthy young woman, married about ten months, and expecting her confinement, was seized with some pains in the lumbar region. On examination, the os uteri was found to be three-fourths dilated. As the pains showed no signs of returning, her physician left her. He was suddenly called to her in about six hours, and then found that the head of the child had been wholly expelled during the profound sleep of the mother. In a moment the body was delivered, and the placenta followed it, the uterus contracting with scarcely any pain. The patient said she had dreamed something was the matter with her, and awoke with a fright, probably at the instant that the head was expelled.

Notwithstanding these cases, it is in the highest degree improbable that any primiparous woman would be delivered during *ordinary sleep* without being aroused and brought to a sense of her condition; perhaps she would be aroused by a desire to relieve her bowels. Before accepting any such account, a statement of all the facts should be obtained from the woman herself. *After* an accident of this kind a woman cannot be ignorant of her own delivery (unless she faints, which is not uncommon). Many a woman who has raised this defence in cases of child murder or infanticide has alleged that she was **unconscious of her pregnancy,** and thus has attempted to excuse herself for not having prepared the articles necessary for childbirth. It is possible that a woman, especially one who is pregnant for the first time, may not be aware of her pregnancy in the early stage, but it is rare for one to advance to the full term without being conscious of it. Women who have borne children, however, have sometimes taken medical advice, and, although nearly at full term, they have not been conscious of their condition. In most cases it may be assumed that a pregnant woman must have some reason to *suspect* her condition; and if only a suspicion existed in the mind of one who did not contemplate the destruction of her offspring, assuredly there would be many circumstances forthcoming which would at once establish her innocence. If this remark applies to married women, it applies with still greater force to those who are unmarried, since the fact of illicit connexion and the fear of its consequences must render them peculiarly sensible of all those changes which take place in the female system during pregnancy.

(*c*) **Suffocation from clothes, and by Urine, Fæces, Blood, Liquor amnii, Mucus, etc.** An infant is easily destroyed by suffocation. If the mouth and nostrils are kept covered for a few minutes, by the face

being closely wrapped in clothes, asphyxia may occur without convulsions or any other marked symptoms (Vol. I, "Asphyxia"). A suspicion of murder may arise in such cases; but the absence of marks of violence, with an explanation of the circumstances, will rarely allow the case to be carried beyond an inquest.

The simple pressure of the clothes or absence of air is not likely to leave any marked traces externally; but internally there may be evidence of asphyxia.

If the other substances mentioned have caused suffocation in a living child, they will be found in the nose, mouth, lungs, or stomach (*vide* Vol. I, pp. 521, *et seq.*); their presence is suggestive of live birth, and conclusive of life during birth, with efforts at respiration; their absence by no means negatives this form of death. Obstruction of the air-passages by mucus and other matters is a frequent cause of death in new-born children. Among twenty-seven children dying in labour, or shortly after birth, eleven died as a result of obstruction of the air-passages with such matters. Eight were born dead, and of those which were alive at birth none survived the first day. In ten of the cases the obstruction was produced by a greenish or greenish-brown slimy mass (meconium and mucus) filling the larynx and windpipe. In two of the cases, in which the child died during delivery, air was found in the lungs, and in only one of these had the air been derived from the act of respiration during birth.

Braxton Hicks has shown that a child may be born living without breathing, simply owing to the **spasm of the larynx** and retraction of the tongue (*vide infra*, p. 176).

Many children are born in similar circumstances when no assistance is available. Cases of this kind, however, rarely give rise to charges of child murder or of infanticide, as no air is found in the lungs, and there are no marks of violence on the body. A child might be killed during delivery by pressure applied to the chest, which might not produce any indication of violence. If the child had not breathed, there would be nothing to show the cause of death; but if air had entered the lungs, then the usual appearances would be found in these organs. In dealing with a case of this kind, it should be remembered that a child with its head born, but detained in the outlet by the size of its shoulders, might die from pressure exerted on the chest by the vagina. It might have breathed, but be born dead with the marks of suffocation about it.

Another condition which may accidentally cause the death of a new-born child during delivery is where the **membranes (or caul)** are carried forward over the head and face, and the act of breathing is thereby mechanically prevented. If no assistance is at hand, the child, although born living, will die soon after birth in consequence of the prevention of respiration. If, when the dead body is found, the membranes are no longer there, the cause of the prevention of respiration would not be apparent. The child, although born living, would probably be pronounced to have been born dead. The delivery of a child with a mask or caul around its head is not an infrequent occurrence.

A mature and healthy child so born was allowed to die by those who had access to it. The caul was simply not removed, so that breathing could not be set up. The lungs contained no air. There was congestion of the brain and lividity of the body, but no mark of violence. There was some evidence that the child had been

born living, and that the cause of death was the prevention of respiration by omission to do that which was necessary. As the medical evidence showed that the child had not breathed, the coroner decided that it had never had any (legal) existence, and that there was no ground for further investigation.

A primiparous female may be ignorant of the necessity for removing the membranes; and thus the child may be suffocated without her having been intentionally accessory to its death. In such cases, however, there would be no marks of violence on the body, or, if present, they would be of such a nature and in such a situation as to be readily explicable.

A case has been reported in which a child was left in its complete membranes for 45 minutes and yet survived by means of artificial respiration.[1]

(*d*) **Prolapse of the Cord.** This is by no means uncommon as a cause of death. The cord is liable to be compressed by the fœtal head and the interference with the circulation of oxygenated blood leads to a condition of asphyxia in the fœtus. Unless the birth is rapid fœtal death must occur. It is not probable that there will be any evidence of its having occurred, but as it may have caused vagitus uterinus or vaginalis, it is possible that by finding vernix caseosa or liquor amnii in the mouth and lungs its occurrence may be suggested; in breech presentations the cord is very liable to be compressed in the absence of skilled assistance.

(*e*) **Strangulation by the Cord.** The cord may be twisted firmly round the neck, and it has no doubt caused death in many instances, even with skilled assistance. The cord is so soft that usually no abrasion of the skin is caused by it, though the actual indentation of the tissues may be apparent: still, even these may be absent, and if the accident is alleged it cannot be proved that it did not occur, though evidences of other violence may show that it was an unnecessary accessory to death.

Pressure on the navel string during parturition produces asphyxia in the fœtus, and careful examination will usually show Tardieu's ecchymotic spots on the surface of the pleura, the thymus gland, the heart and pericardium.

There is a singular cause of death in reference to the umbilical cord which must here be noticed. Cases have been reported from time to time in which an actual knot has been found in the cord. For such a knot to occur the body of the child in its movements *in utero* must have passed through a loop of the cord, forming a knot, which may be tightened by its further movements, or remain loose until delivery; the fœtus thereby unconsciously commits suicide by compressing the vessels and arresting all circulation between it and the placenta. The fœtus may perish before birth, or it may die from the compression naturally produced by a protracted labour. Such an accident may occur by the fœtus passing through a loop while its head is passing through the mouth of the uterus, so as to form a knot at the very moment when the body passes into the world. Whether the child had breathed effectively or not, it might die by fatal compression of the cord before its birth was completed. The cause of death would always be apparent if the cord could be obtained for examination. The cord may be the means of producing other marks of injury on the body of the fœtus *in utero* which

[1] *B.M.J.*, 1910, 1, p. 1289.

however, could hardly be mistaken for the effects of violence in the extra-uterine state. The amputation of the limbs of the fœtus has been produced by a tightly constricted cord, but such an occurrence must be extremely rare.

(*f*) **Hæmorrhage from the Cord.** In precipitate labour the cord may be torn, and it is usually assumed that such tearing will prevent hæmorrhage, but as to the truth of such assumption the evidence seems to be inconclusive. It may, however, be severed in other ways, either by accident or deliberately by the mother, and she may be unable to tie it herself; it is thus possible for a fatal bleeding to take place if skilled assistance be not at hand, and several such cases are recorded.

Bleeding from the cord has been observed to take place at various periods after birth, and has led to the death of the child. Death from bleeding may be commonly recognised by the blanched appearance of the body and a want of blood in the internal organs. There are several cases on record in which the cord was ruptured close to the abdomen without causing the death of the child. Bleeding is more likely to prove fatal when the cord is divided by a sharp instrument than when it is lacerated; and its dangerous effects on a child are likely to be great in proportion as the division is made near to the navel. It has been described as a case of infanticide by *omission*, when a self-delivered woman neglects to apply a ligature to the cord in these circumstances: because it is said, she ought to know the necessity for this in order to prevent the child from dying from hæmorrhage. Such a view assumes not only malice on the part of the accused, but also that in the midst of her distress and pain she must necessarily possess the knowledge and bodily capacity of an accoucheur—a doctrine wholly repugnant to the common feelings of humanity. This question was, however, raised in the case of *R.* v. *Dash*. There was no doubt that the child had breathed, and that its death had been caused by bleeding from the lacerated umbilical cord. The medical witness admitted that the cord might have been torn through by the mere weight of the child during labour; and the jury acquitted the accused mother on the ground that she might have been ignorant of the necessity for tying the cord or might not have had the power to tie it. The cord, especially when short, may become accidentally ruptured during delivery.

A child was born alive after a strong pain; and on examination it was found that the cord was torn through at about an inch from the abdomen. The cord was only four inches and a quarter in length. In another case there was no rupture but great pain caused to the woman during delivery; and the cord was only five inches long.

Bleeding from the vessels of the navel-string may prove fatal several days after birth, even when a child has been properly attended to, and the navel-string has separated by the natural process.

(*g*) **The Effects of Abnormal Presentations, Placenta Prævia or Premature Separation of the Placenta.** Without skilled assistance such conditions are very likely to prove fatal to both mother and child.

Immaturity or Debility

A child may be born either prematurely or at the full period and not survive its birth, owing to a natural feebleness. This is very

commonly observed among immature children. Such children may continue in existence for several hours, breathing freely, and may then die from mere weakness. These cases may be recognised by the immature condition of the body and the general want of development.

There will also be the negative evidence of no other possible cause of death. The importance of prematurity as a contributory or sole cause of neo-natal death has already been stressed.

Malformations Incompatible with Life

These are very numerous, and should be divided into those which are incompatible with life for more than a few minutes or hours, and those which, unless removed by surgical aid, will prove fatal after the lapse of a few days or longer.

To the former class belong **acephalous and anencephalous monsters, extroversion of the heart,** and certain abnormalities of the main blood vessels ; to the latter belong **imperforate anus, constriction of œsophagus or duodenum or obliteration of part of the alimentary canal.** The cases are too numerous to need references, but there can be no difficulty in determining whether they are such as to account for death. There is no liberty to destroy monstrous births ; and the presence of marks of violence in such cases should be regarded with suspicion. It is the more necessary to make this statement, as there is an idea among the vulgar that it is not illegal to destroy a monstrous birth.

The only case on this point is that of two women who were tried at the York Assizes early in the last century for drowning a child born with some malformation of the head, in consequence of which it was probable that it could not survive many hours. There was no concealment on the part of the accused, who were not aware of the illegality of the act. The fact that the offspring is not likely to live more than a few hours does not justify premature destruction.

Spasm of the Larynx

Strong and healthy children may die from non-inflation of the lungs. They are born alive, and on coming into the world make attempts at inspiration, but, as Braxton Hicks has pointed out, owing to spasm of the larynx and retraction of the tongue, the air is unable to enter the lungs. The child dies, and as no air is found in the lungs, the child is wrongly pronounced to have been born dead. A careful inspection of the fauces may show the presence of mucus or meconium, or a condition of the epiglottis which may account for non-respiration.

Diseases Acquired in Utero

The discovery of any of the fœtal organs in a morbid condition amounts to nothing unless the disease has advanced to a degree which would be sufficient to account for the child's death. There are, doubtless, many obscure affections, particularly of the brain, which are liable to destroy the life of a child without leaving any well-marked changes in the dead body. Apoplexy and asphyxia are the usual causes of death among new-born children, the latter the more common. Probably diseases of the lungs are of the greatest importance in a medico-legal point of view ; because, by directly affecting the organs of respiration, they render it impossible for a child to live, or to survive its birth for a

long period. The diseases in the fœtal state are principally hepatisation and syphilis of the lungs—the existence of which it is not difficult to discover. They render the lungs heavier than water, and prevent them from acquiring that buoyancy which in a healthy state they are known to possess. It is not common to find the lungs diseased throughout—a portion may be sufficiently healthy to allow of a partial performance of respiration.

Reference must also be made to erythroblastosis fœtalis, due to Rh-factor incompatibility between mother and fœtus, as a cause of still-birth and neo-natal death. Unexplained hæmorrhages in the lung, or into the suprarenal glands or into one or other of the body cavities may also be responsible for death.

In addition to diseases thus leaving traces in the internal organs, it must not be forgotten that any of the acute specific fevers in the mother will almost certainly lead to the expulsion of the contents of the uterus, as will maternal syphilis, chronic renal disease, toxæmias of pregnancy, etc. If the mother's attack be at all severe, the child is nearly sure to be born dead from a toxæmia of its blood, or sometimes even from an actual and definite invasion of the child by the microbes of the disease with definite skin eruption. Small-pox and scarlet fever rashes have been thus definitely seen on a new-born child.

Disease of the Placenta

The changes found in the placenta which may be assumed to have caused the death of the fœtus are as follows[1]:—

Retroplacental hæmorrhage, or hæmatoma, as evidenced by clots of various age, or by the depressions on the maternal surface caused by them.

Intraplacental hæmorrhages.

Extensive white infarction.

Extensive red infarction, found almost always in association either with retroplacental hæmorrhage or toxæmia of pregnancy, or both.

Syphilitic changes in the villi.

An unusually small placenta.

Absence of one artery from the umbilical cord associated with absence of the corresponding hypogastric artery of the fœtus.

Some of these changes are sufficient to kill the fœtus by their direct action, *e.g.*, retroplacental hæmatoma ; others, such as syphilitic changes in the villi or red infarction, are indicative of other lethal conditions.

The changes in the placenta attributable to syphilis are by no means easy to detect. Naked-eye examination reveals no specific change, and the principal histological changes are as follows :—

(1) A uniform increase in the size of the villi, resulting in their being more crowded together than normally and implying a corresponding reduction in the size of the intervillous spaces. This change is due to an increase in the amount and density of the stroma, the collagen fibres being more numerous and the cells more abundant and closely packed.

(2) A greatly diminished vascularity of the villi. The blood vessels are usually absent ; when present they are greatly diminished in number and size. The walls of the vessels, when present, are not altered.

[1] Holland, Report No. 7, Ministry of Health, 1922, p. 106.

These changes sometimes affect the whole placenta uniformly, and sometimes only partially. The villi coming from one or more main chorionic stems may be affected, whilst others escape, healthy and changed villi being seen side by side in the same section. It is obviously absurd to diagnose syphilis from the discovery in a section of a few isolated large, dense, avascular villi. Furthermore, it is obvious that little assistance may be obtained from one section; either one very large section or several small sections must be used.

The only absolute proof of syphilis in the placenta is the demonstration of spirochætes.

WHETHER OR NOT THE CHILD WAS BORN DEAD

The answer to this question will depend upon four factors, viz. :—

The period of development to which it has reached.
The presence of ante-partum *rigor mortis*.
The presence of ante-partum decomposition.
The absence of evidence of live birth.

Any one of the first three factors may give positive evidence; the fourth is of doubtful value.

Stage of Maturity

One of the first questions which a medical witness has to consider in a case of alleged child murder or infanticide is that which relates to the age or probable degree of maturity to which the deceased child may have attained *in utero*. The reason for making this inquiry is, that the chances of natural death, at or before birth, in all new-born children are great in proportion to their immaturity; and that, assuming them to have survived birth, the signs of respiration are usually obscure. The greater number of children who are the subjects of these investigations have reached the eighth or ninth month of gestation; yet charges of murder or infanticide might be extended to the wilful destruction of children at the seventh month or under, provided there is clear evidence of life after birth.

In order that a child may become the subject of a charge of murder or of infanticide, the law of England does not require that it should be born *viable*, *i.e.*, with a capacity for continued life. A child may be born alive at the sixth or seventh month; but because it is much less likely to survive than one born at the eighth or ninth month, this is not a ground of exculpation for any person who wilfully destroys it. The real question does not refer to the period of gestation at which a child may be born, but to the fact of its being *living* and *entirely born* at the time when it was destroyed. The meaning of the term *viability*, as applied to new-born children, has been elsewhere considered ("Shortened Gestation" and "Live Birth," pp. 33 and 136, *et seq.*). It means simply the ability to continue life. The crime of child murder or infanticide implies the destruction of a new-born child "born living," whatever may be its state of development, shape, strength, or capacity to live. Child murder or infanticide is therefore entirely independent of the question of viability, and yet a medical witness is sometimes asked—Was the child viable? This question is put in order to show how far the strength of the child would enable it to resist the violence suffered.

Children cannot be destroyed with impunity merely because they happen to have been born under the seventh month, neither can a child be assumed to have been born dead, and an inquiry into the cause of death dispensed with, unless it can be medically established that it has passed the seventh month of gestation.

As to children born at the fourth or fifth month of gestation, a charge of concealment of birth may arise. In these circumstances it is not necessary to prove that it was born living. At the same time, as such births at the fourth and fifth months are always the result of abortion either from natural or criminal causes, the charge is generally merged in the criminal offence of procuring abortion. Here, again, it is not required to prove by medical evidence that the aborted fœtus was living when expelled from the womb. In nearly all cases of child murder or of infanticide, it will be found that the child has passed the seventh month of utero-gestation.

Ante-partum Rigor Mortis

This condition has been recorded by several observers, and has even been used as a test of degree of maturity. The following report shows that it is not so very infrequent, but is probably overlooked. Its occurrence proves that the child was born dead.

Rigor mortis in the fœtus *in utero* was the cause of difficult labour. The patient was a primipara, aged thirty-seven ; the os was fully dilated but no advance of the head had taken place for several hours. On applying forceps considerable resistance was experienced. Progress was slow until after securing extension, the head came down well. There was further difficulty in extracting the body, which gave the impression of being much too large for the maternal passages. The fœtus, in fact was not large, but rather under the average. The difficulty had arisen at the various stages : first, the normal flexion had not taken place, and at a later stage the stiffness of the neck had prevented extension, and the lateral flexion of the body had been interfered with owing to the same cause. *Rigor mortis* was well developed all over the body, the arms and legs being stiff and in the flexed position. On balancing the child's body on one hand its back kept almost straight and the legs in the same flexed position, the heels not dropping below the nates. Owing to the forcible flexion of the neck the head moved upon the trunk like a hinge joint. At the moment of applying pressure with the forceps there was a crunching noise quite audible, sounding as if one were breaking a bone.

For further references, *vide* Vol. I., " Rigor Mortis."

Signs of Putrefaction in Utero

The phenomena of putrefaction in air have been elsewhere described (Vol. I., pp. 197, *et seq.*) ; but the changes which ensue when a child dies and is retained within the uterus may be briefly adverted to, because they may sometimes form a subject for judicial inquiry. According to Devergie, when a child dies *in utero*, putrefaction takes place as rapidly as in the open air, but this is incorrect.

In an advanced state of *uterine putrefaction* (intra-uterine maceration), the body of the child is so flaccid that when placed on a table it becomes almost flattened by the mere gravitation of its parts. The skin is of a reddish-brown colour—not green, as in a putrefied body exposed to air. The cuticle covering the feet and hands is white, and sometimes raised in blisters, filled with a reddish-coloured serum ; the bones are movable, and readily detached, from the soft parts. In the

opinion of Devergie, the principal difference between uterine and atmospheric putrefaction, in the body of a new-born child, is seen in the colour assumed by the skin; but it must be remembered that, should the child remain exposed to the air after its expulsion, the skin may acquire the colour observed in cases of atmospheric putrefaction. There is also an easily recognisable (once it has been experienced) smell about intra-uterine decomposition, quite peculiar and pathognomonic; it is rather a sickly than pungent smell of decomposition. The changes which have just been described are such as we may expect to find when a child has been retained in the womb eight or ten days after its death. When it has remained for some weeks in the uterine cavity, the body has occasionally been found in an adipocerous state, or even encrusted with phosphate of calcium. If in any case we are able to state distinctly that the body of a child has undergone uterine and not atmospheric putrefaction, it is clear that it could not have come into the world alive, and no question of murder could arise. In ordinary putrefaction in air, a child may have been really brought into the world living, and the process may have destroyed every proof of that fact.

Where a child has died *in utero* twenty-four hours before it was born; and is examined soon afterwards, there will be no marks of putrefaction about it, unless the membranes have been ruptured, and the appearances will closely resemble those met with in the body of a child that has been born alive, and died without breathing; or of one that may have died in the act of birth. It will be impossible to say, in such a case, whether the child came into the world living or dead. The dead fœtus retained *in utero*, with the membranes unruptured, undergoes one of three changes—maceration, putrefaction, or mummification. The first is the most common condition, but the changes differ from those which take place in the body when exposed to air. Putrefaction, in its common signification, is rarely met with.

After a child is born and death takes place and the body is exposed to the air, changes of putrefaction elsewhere described (*vide* Vol. I, pp. 197, *et seq.*) take place similar to those which take place in the dead body of an adult. The body of a child cools more rapidly than that of an adult, and putrefies, *cæteris paribus*, also more rapidly, and inasmuch as new-born babies are frequently thrown into water, cesspools, etc., attention should be drawn to those agencies, as they have an effect upon decomposition.

But, because of the absence of bacteria from the respiratory and alimentary passages of an infant who has not breathed or fed, decomposition is often very much delayed, and the occurrence of mummification is favoured, especially when the body is kept in a box or drawer, or elsewhere if the atmosphere is warm and dry.

Read states that he met with a case of desquamation of the cuticle in a living new-born child. The woman was prematurely confined, in consequence of an accident. The hands and feet of the child when born were denuded of cuticle, which hung from them in shreds. The child was apparently at the eighth month, and lived twenty-four hours.[1] This, at any rate, shows that desquamation of the cuticle in a new-born child is not always a sign that it has been dead for a considerable length

[1] *Amer. Jour. Med. Sc.*, October, 1861, p. 583.

of time. All the other signs indicative of putrefaction would, in such a case, be absent, and from this fact a medical witness would be able to draw a clear distinction.

In certain cases, where the body of a child has been long buried in the earth, the bones only may be producible. The question that would be likely to arise here would be :—Whether the bones were those of a new-born child or of one that had survived its birth for some weeks or months. There will be no difficulty in coming to a conclusion on the question (see Vol. I, "Age"), and the answer may at once put an end to the charge of infanticide.

Negative Evidence from Absence of Breathing, etc.

When negative results are given by the tests already described for live birth, the value of such negative evidence is small. But there are certain generalisations which may usefully be stated here, viz. :—

1. That a child may be born alive and be criminally destroyed before it has breathed.

2. That there may be no certain medical signs by which a child which has not breathed can be proved to have been living when it was subjected to violence.

3. That a new-born child may be destroyed accidentally or purposely by the prevention of respiration during delivery.

4. That a child may breathe before, during, or after birth, but the hydrostatic test will not enable us to say with any degree of certainty, at which of these periods the act of respiration was performed.

5. That the proof of respiration shows that the child has *breathed*, not that it has been *born alive*.

6. That by taking together the colour, volume, consistency, appearance of developed air-cells, absolute weight, and buoyancy of the lungs, we may be able to state whether the child has or has not breathed, but this docimasia pulmonum does not always enable us to determine with certainty whether a child has been born living or dead, or has died a violent death.

7. That if the lungs are fully and perfectly distended with air by the act of breathing, this affords a strong presumption that the child has been *born alive*, since breathing during birth is in general only partial and imperfect.

8. That when lungs have undergone perfect respiration, the air cannot be expelled by compression of the divided parts, so as to cause them to sink.

9. That the lungs of children that have lived after birth may *sink* in water, owing to their not having received air, or to their being in a diseased condition, but such a result is no proof that the child was born dead, for a child may live for a time when only a portion of the lung has received air.

10. That the lungs of children which have not breathed and have been born dead may float in water from putrefaction, or from artificial inflation.

11. That the lungs as situated in the chest undergo putrefaction very slowly ; that, if but slightly putrefied, the air may be easily forced out by

compression; and if much putrefied, either the case must be abandoned or other sources of evidence sought for, for neither the putrefaction nor the sinking proves that the child was born dead.

12. That while lying in the chest, the fœtal lungs are not easily inflated, and that the difficulty in inflating them is great in proportion as the child is immature.

13. That lungs artificially inflated while in the chest usually resemble those organs in which respiration has been only imperfectly established.

14. That when the lungs are very heavy, and contain but little air, it cannot with certainty be inferred that respiration has been established. The facts, *cæteris paribus*, may be explained by supposing that the lungs were naturally heavy, and that they have been artificially inflated.

15. That certain changes in the umbilical vessels, and the separation by a vital process and cicatrisation of the umbilical cord, indicate live birth.

16. That the open or contracted state of the foramen ovale or ductus arteriosus furnishes no evidence of a child having been born alive. These parts may become closed and contracted *before birth*, and therefore be found closed in a child born dead; or they may remain open after birth in a child born living, even subsequently to the establishment of respiration.

17. That the absence of meconium from the intestines and of urine from the bladder are not proofs that a child has been entirely born alive, since these liquids may be discharged during the act of birth.

18. That the presence of farinaceous or other food in the stomach, or of foreign substances swallowed, proves that a child has been entirely born alive.

19. That the presence of blood, meconium, vernix caseosa, or the discharges in the stomach and air-passages, does not necessarily prove that a child was born alive.

On the question of the length of time during which the child lived after birth, the following observations are in point:—

(1) The period for which a new-born child has survived birth cannot be determined by any certain sign for the first twenty-four hours.

(2) After the said period an inference may be drawn from certain changes which take place progressively in the skin and umbilical cord externally, and in the viscera on inspection; such changes allow of only an approximate opinion within the first five or six days.

(3) The contraction of the ductus arteriosus, and the closure of the foramen ovale, take place from natural causes at such uncertain intervals as to render it difficult to assign a precise date of survival from the condition of these parts.

If a child is destroyed either during birth or within a few minutes afterwards, there will be little medical evidence to indicate the period at which its destruction took place. It is most probable that in the greater number of instances a child is destroyed either during birth or more probably immediately afterwards; and, therefore, the conditions above described are rarely available in practice. If any exception be made, it is with respect to the nature, situation, and extent of marks of violence; but the presence of these depends on mere accident.

Although medical evidence can usually show, from the state of the lungs, that a child has breathed, it is rarely possible to prove, in a case of alleged child murder or infanticide, that life certainly continued after birth. We could venture upon this inference only when the signs of breathing were full and complete, or when some article of food was found in the stomach. The conclusion to be drawn from these observations is, that if the Court were to insist upon conclusive medical evidence of entire live birth in every trial for child murder or infanticide, there would be very few convictions, except where a confession was made by the accused, or the crime was committed in the presence of eyewitnesses.

The courts will admit evidence of life in a child before the establishment of respiration. In *R.* v. *Brain*,[1] the judge said that a child might be born alive, and not breathe for some time after its birth. In *R.* v. *Sellis* it was alleged that the accused had murdered her child by cutting off its head. The judge directed the jury that if the child was *alive* at the time of the act, it was not necessary, in order to constitute murder, that it should have breathed. Breathing is regarded as only *one* proof of life; any other kind of evidence showing that a child had lived will be received.

A medical practitioner must first prove that the child under examination has recently died, or that there are good grounds for believing it to have been *recently living.*

VIOLENT OR CRIMINAL CAUSES OF DEATH

We have now to consider those causes of death which are independent of the existence of congenital disease or other natural causes. There are certain forms of child murder or of infanticide which are not necessarily accompanied by appearances indicative of violence: these are suffocation, drowning, exposure to cold, and starvation. The evidence in many of such cases is of the same nature as that which will be looked for in the case of an adult. The said causes of death will be considered in the following order:—

Suffocation.
Strangulation.
Cold and exposure.
Starvation.
Violence by wounds internal and external.
Poisoning.
Drowning.
Deliberate neglect to tie the cord.
Burning.

Suffocation

This is a common cause of death in new-born children (for overlaying of living infants, *vide* Vol. I). A cloth may be placed over the child's mouth, or thrust into this cavity, either during birth or afterwards, and before or after the performance of respiration. To the latter case only could the term suffocation be strictly applied. A child

[1] 6 C. & P. 349

may be thus destroyed by being allowed to remain closely compressed under the bed-clothes after delivery, or by its head being thrust into straw, feathers, dust, ashes, or similar substances. The appearances in the body are seldom sufficient to excite a suspicion of the cause of death, unless undue violence has been employed. There is commonly merely lividity about the head and face, with slight congestion of the lungs. A careful examination of the mouth and throat should be made, as foreign substances are sometimes found in this position, affording circumstantial evidence of the mode in which the suffocation has taken place. Thus **wood, straw, feathers, dust, tow, or a hard plug of linen** may be, and in some cases have been, found blocking up the mouth and fauces, drawn into these parts by aspiration when the mouth of a child has been covered with such substances (see "Smothering," Vol. I.). If a child has lived sufficiently long to be fed, it may be accidentally suffocated by the entrance of portions of **food** into the windpipe and air-passages.

An infant, thirty days old, was found dead in bed. The mother stated that the child was healthy, and was put to bed after having sucked well at 7 p.m. the previous evening; also once in the night about 2 a.m. On awaking at 4.30 a.m. she found the child dead. It was lying at her right side—the farthest side from the father, and on its own left side looking towards its mother. When examined it was on its back; the hands were clenched, the lips blue and pouting, and the tongue thrust out a little way between them. There were no marks of violence or flattening of the features. The lungs and right cavities of the heart were distended with blood. The stomach contained a quantity of curdled milk. Upon opening the larynx a small quantity of the curd of milk was found resting on the vocal chords of the larynx and lining the upper two or three rings of the windpipe, thus completely blocking up the tube, which in calibre was not larger than a goosequill. It appeared that on the previous day the child had frequently thrown up its milk; it had probably vomited it in the night while its face was turned towards the pillow, and had by aspiration drawn a portion of the curd milk into the air-passages, and thus caused death by suffocation. An infant of this age would not have the power to relieve itself in such a position.

A new-born child may be suffocated by having its head held over **noxious vapours** such as coal gas or burning sulphur, the poisonous vapours of chloroform, ether and similar substances.

On the other hand, even if it be clearly proved that death has been caused by suffocation, the suffocation might have been accidental (*vide* Vol. I, "Suffocation").

Sometimes the body is found maltreated, with severe fractures or contusions on the skull, and marks of strangulation on the neck; concealed in a feather bed or privy; or cut up and burnt. Such violence may excite suspicion of murder and suggest that the allegation of death from accidental suffocation is a mere pretence. Real cases of death from accidental suffocation, when properly investigated, can never involve an innocent woman in a criminal charge, although the facts may show in some instances that the death of the child was really due to great imprudence, gross neglect, or culpable indifference on her part. Thus a woman knowing or having reason to believe that her delivery is impending is secretly delivered at night. The child is born under the bed-clothes; no effort is made to remove it, and it dies from suffocation. In some cases of this kind a woman may be unable to make the necessary exertion to uncover the head of the child so as to allow it to breathe. In others, however, there is no desire to save life, and the child is found dead.

The following case shows that in England, even when the evidence for the prosecution is very strong, the circumstances will be most favourably interpreted towards the accused.

In *R.* v. *Mortiboys* the body of the child was discovered, lying on its abdomen, in a box containing wool, with its face raised and its mouth open. A red worsted comforter had been passed twice around the neck, and was tied a second time in a single knot over the chin. In the mouth, which was open, was found a small quantity of fine flecks of wool. The medical evidence showed that the child had been born alive, the left lung being fully inflated. The brain was congested. There was no mark produced by the ligature on the neck, either externally or internally. Death was referred to obstructed respiration (suffocation), caused partly by the ligature and partly by the wool in the mouth; but the latter was considered to be the more active cause. The judge said that if the accused had intended to choke the child with the wool, she would have inserted enough to fill its mouth. The accused was acquitted.

In another case the child was suffocated by a quantity of mud having been forced into its mouth and throat. The presence of mud in the gullet was incompatible with its having entered by gravitation. In another case several small pieces of straw were found in the stomach of a child, similar to those which were in the bed where the birth took place. In another case it was found that a mass of dough, or bread-pulp, had been forcibly impacted in the throat and larynx of the child, and it was found to be accurately moulded to the parts. In another case a plug of flax was discovered in the mouth.

Any foreign substance found in the air-passages should be most carefully examined. The following report illustrates the need for caution in such cases.

The body of a new-born child was found in a marshy soil, and from an examination it was stated that it had breathed, had died from suffocation, and had been buried where the body was found. This opinion was based chiefly on the discovery of a dark-coloured substance in the windpipe and bronchial passages, which was pronounced to be earth from the marsh in which the body was buried. The mother said that she had been suddenly delivered over a tub, used for the purpose of a privy, that the child fell into the excrementitious fluid, and that she afterwards removed the body and buried it in the place where it was discovered. Her story was corroborated by the discovery that the dark substance present in the air-passages and in the gullet was not part of the earthy soil in which the body had been buried, but dried fæcal matter. This discovery of the real nature of the substance exonerated the woman from the charge of murder.

A servant girl gave birth to a healthy child which was found alive about a quarter of an hour afterwards in a privy. It lived for a few minutes. Its jaw was broken, its cheek torn, and the mouth contained ashes, some of which were found in the back part of the throat. The body was blanched, and there had evidently been a great loss of blood from the wounds and the torn umbilical cord. There was no engorgement of the lungs, nor any subpleural ecchymoses. The lining membrane of the trachea was stained with ashes, and a small cinder was found in the left bronchus. There was no question respecting live birth, as the child was living when found, but questions arose as to the cause of death, and whether death was accidental or the result of violence wilfully applied after birth. The mouth of the child had been forcibly torn open and filled with ashes in order to suffocate it. The ashes must have been drawn by aspiration into the air-passages, and death was caused partly by suffocation and partly by haemorrhage from the wounds, the child's body being bloodless. The condition of the lungs was not inconsistent with death from suffocation.

Sometimes living children are killed by suffocation in the act of birth; death being caused not by the actual infliction of violence, but either through accident or design by preventing the performance of that act which is necessary to maintain life after the child is born.

A pregnant woman, thinking she was about to have a motion, sat on an earthen pitcher, two feet in depth, which happened to be full of water. She was there delivered of a child, which fell into the water, and was thus prevented from breathing. The child was full grown, and its body was free from putridity. It weighed six pounds, and measured twenty inches in length. There were no external marks of violence, and the navel-string had been *tied*. The lungs weighed two and a half ounces; they were of a liver colour, contained no air, and sank in water. The medical opinion was that, from the size and general appearance of the child, and the state of the parts discovered on dissection, it was mature, that it had not breathed, and life might have been either wilfully or accidentally destroyed. The woman was not prosecuted, probably because the death of the child might have been accidental. There was no medical proof that the child had been born alive, although it seemed highly probable that its life had been destroyed in the act of birth.

Post-mortem Appearances in Smothered Infants. These are fully described, in reference to adults, Vol. I, p. 523; and they are similar in new-born children, provided respiration has been fully performed. Great importance is to be attached to the discovery of subpleural or punctiform ecchymoses on the lungs of children; and also to small effusions of blood on the surface and in the substance of the thymus gland. If the lungs float in water, as the result of breathing, then the appearances described may be met with; but in three instances Tardieu met with similar appearances in children whose lungs had not received air, and sank when placed on water. They were children prematurely born, and in conditions which prevented full vital development. The subpleural ecchymoses met with in children in these circumstances were ascribed by Tardieu to the efforts made to breathe after birth. They are, however, perhaps more commonly produced during parturition by obstruction to the fœtal circulation brought about by pressure on the umbilical cord, and consequent asphyxia.

Much has been written touching this *post-mortem* appearance in death from suffocation in new-born children. The mistake is made of assuming that subpleural ecchymoses indicate the *cause* of the asphyxia; they, of course, do nothing of the sort. They suggest that the person in whom they are found died from asphyxia, but do not indicate the cause.

Strangulation

Strangulation is a common means whereby a new-born child is killed. Sometimes, however, strangulation occurs accidentally by the twisting or coiling of the umbilical cord around the neck of the child while it is in the womb (see p. 174), or during delivery. We must not hastily conclude, from the red and swollen appearance of the head and face of a child when found dead, that it has been destroyed by criminal strangulation. There is no doubt that errors can easily be made with respect to this appearance. When a child's head or face looks swollen, and is very red or black, the uninstructed are liable to conclude that the child must have been strangled. But those who practise midwifery know that there is nothing more common in natural births, and that the swelling and deep colour gradually disappear if the child lives but a few hours or days. This appearance is particularly observable in those cases where the navel-string happens to be twisted around the child's neck, and where the head is born some time before the body.

Strangulation by the Navel-string. Strangulation by the navel-string can, in the medico-legal sense of the term, refer only to those cases in which it becomes firmly twisted around the neck *after* the respiratory process has been established. This is a rare occurrence, because in these circumstances death usually takes place by compression of the cord, and by the consequent arrest of circulation before the act of breathing is performed. The internal appearance met with in death from this causeis a congested state of the cerebral vessels, and ecchymotic spots on the surface of the heart, lungs, and thymus gland. The presence of ecchymosis on the scalp, as well as of lividity of the face, is very common in new-born children when the labour has been tedious and difficult ; and therefore, unless there were some distinct marks of pressure about the neck, such appearances would not justify any suspicion of death from strangulation.

It has been suggested that strangulation from the wilful application of any constricting force to the neck can be distinguished from accidental strangulation caused by the cord, by the fact that in the former case there is a livid or ecchymosed mark or depression on the neck. But to this view it may be objected that such a mark is not a constant accompaniment of homicidal strangulation. Severe violence to the neck commonly produces at the site of constriction not only ecchymosis, but a laceration of the skin, muscles, and windpipe ; but these appearances are not always found.

In a reported case where a new-born child had been killed by strangulation, great violence had been used, but there was no trace of discoloration in the course of the ligature, or of ecchymosis in the tissues beneath. The muscles compressed were very dark in colour. The skin had been so compressed as to give the impression that coarse towelling of a close texture had been used.

In most cases when a ligature is applied during life the skin on each side becomes much swollen, and presents an œdematous character. This indicates an application of violence when there is still some vital power in the body of the child. The navel-string itself may be used as a means of constriction, and the mark or depression may sometimes present an appearance of ecchymosis. Among various cases which might be quoted in support of this statement is the following :—

The labour of a primipara was of a lingering kind, owing to the size of the head ; and the child came into the world dead. The navel-string was found coiled three times around the neck, passing under the right armpit ; and upon removing it *three parallel discoloured depressions* were distinctly evident. These extended completely around the neck and corresponded to the course taken by the coils. The child appeared as if it had been strangled.

If this child had been born secretly, and the cord removed, the condition of the neck might have created a strong suspicion of homicidal violence. Strangulation after birth could not, however, have been alleged, because there would have been no proof of respiration.

In another case the cord, which was short, was so tightly twisted around the neck of the child that it had to be divided before delivery could be accomplished. There was a deep groove formed on the neck, conveying such an impression that, in the absence of any knowledge of the facts, medical witnesses would have been prepared to say that the child had been wilfully strangled by a rope.

A more accurate diagnosis might have been formed, as in the preceding case, by examining the state of the lungs.

In another case a child was born dead with the cord tightly twisted around its neck. When the cord was removed, the neck exhibited a livid circle of a finger's breadth, smooth and shining; but on cutting into this mark, no subcutaneous ecchymosis was found.

Ecchymosis in the depression furnishes no distinction between constriction produced by criminal means and that which may result accidentally from the navel-string. In the following case a woman charged with the murder of her child by strangulation appears to have been unjustly condemned.

The child had fully and perfectly respired, the lungs weighed one thousand grains, and, when divided, every portion floated on water, even after firm compression. There was a circular depression on the neck, which was superficially ecchymosed in some parts. This appeared to be a case in which a mark on the neck was accidentally produced by the umbilical cord during attempts at self-delivery on the part of the woman: she was nevertheless convicted, chiefly because two medical witnesses said that a soft and yielding substance like the umbilical cord could *not* produce a depression and ecchymosis on the neck of the child during birth. They attributed the mark to the wilful application of a ligature like a garter; but experiments have clearly shown that the umbilical cord has sufficient strength to produce fatal constriction.

In another instance the navel-string and the membranes were actually used by the woman as a means of strangulation: the child had not breathed, but was thereby prevented from breathing. There was superficial ecchymosis on each side over the muscles of the neck. The medical evidence showed that the cord had been violently stretched, and employed as a means of strangulation. The child had *not breathed*, and the medical witnesses considered that it had been born dead, owing to the violence used by the woman. The cause of death was not strangulation, but arrested circulation.

The case proves that ecchymosis (a blue mark) may be the result of violent constriction produced by the navel-string.

In one case the navel-string, which was of its full length, had been used as a means of strangulation. It was twisted once around the neck, passed under the left arm, over the shoulders, and around the neck again, forming a noose or knot, which, pressing upon the throat, must have caused strangulation, as the tongue was protruded, and there were other clear indications that the child had been strangled. The hydrostatic test applied to the lungs proved that respiration had been performed.

When the mark on the neck is deep, broad, much ecchymosed, and there is extravasation of blood beneath, with injury to the muscles or windpipe, and ruffling or laceration of the skin, it is impossible to attribute these appearances to accidental pressure by the navel-string. The lividity produced by it in the cases hitherto observed has been only slight and partial, and unaccompanied by laceration of the skin, or injury to deep-seated parts.

It has been doubted whether a child can be born and perform the act of respiration fully and completely, with the navel-string so tightly round the neck as to produce great depression of the skin and ecchymosis, *i.e.*, to stimulate homicidal strangulation. It is important, therefore, when this hypothesis is raised in order to account for a suspicious mark on the neck, to examine closely the state of the lungs. Unless the cord is designedly put round the neck of the child *after* the head has protruded, the effect of the expulsive efforts of the uterus, when a coil has become *accidentally* twisted round the neck, would be to tighten it compress

the vessels, and kill the child by arresting the placental circulation, at the same time that this pressure would effectually prevent the act of breathing. Hence the lungs usually present the appearances met with in still-born children; but this state of things may sometimes occur, and a child may breathe, and die strangled by the umbilical cord before its body is entirely born.

A careful examination of the neck may show whether a ligature has or has not been wilfully applied after birth. In examining a suspicious mark round the neck of a new-born infant, it should be noticed whether it does not, by its form or course, present some peculiar indentations or irregularities which may render it certain that some kind of ligature has been wilfully employed after birth. When it is found that a child has fully breathed, the presence of a deeply ecchymosed or an œdematous mark on the neck, with injury to the skin and muscles, is, *cæteris paribus*, presumptive of homicidal strangulation. Death from accidental constriction by the cord during delivery should, as a general rule, leave the lungs in their fœtal condition.

Marks on the neck of a child may be accidentally produced by the navel-string without necessarily causing the death of the child (cf. *supra*) and, indeed, there is much less risk of strangulation from the cord than is commonly believed.

Strangulation by means other than the Cord. The *appearances* met with in the body in death from strangulation have been elsewhere considered (Vol. I, pp. 561, *et seq.*). The facts of a case will serve to show the appearances as they may present themselves in a new-born child :—

A maid-servant was secretly delivered of a child. The body was full-grown, and there was a piece of tape which went twice around the neck, and had been tied tightly in a bow. The tongue protruded between the lips; two deep furrows were found around the neck after the removal of the tape; there was great œdema of the skin between and above them, and the right hand was clenched. The lungs were of a light-red colour: they filled the chest, were highly crepitant, and floated readily on water, even when divided into sixteen pieces, and these had been submitted to strong pressure. They weighed, however, only 626 grains. The heart was healthy; the right side contained some coagula of blood, whilst the left side was empty; the foramen ovale was open. The scalp was much congested, the pia mater was also congested. The inferences drawn from these facts were; that the child had been born alive, and that it had died from strangulation.

A discoloration may be in detached spots or patches—situated in the fore part of the neck, and evidently not arising from the employment of any ligature. These marks may depend on the forcible application of the fingers to the neck of the child, and the indentations have been known to correspond—a fact which has at once led to a suspicion of the cause of pressure and the mode of death. Impressions of nails or fingers on the neck do not necessarily imply that they have been caused by an attempt at strangulation. Accident during self-delivery may lead to their production. At the same time it should be borne in mind that a superficial mottling of the skin occurs after death in new-born infants, in parts where moderate pressure only may have been accidentally made. This would not be attended with ecchymosis, and its true nature would be at once determined by comparing the discoloured spots with the surrounding skin. It may be alleged that such marks have been

accidentally made, *e.g.*, by the forcible pressure produced by the child's head during labour, an explanation which is highly improbable, if respiration has been performed—although a child has been known to breathe in breech-presentations, while the head was still in the vagina. They will be more commonly referred to a violent attempt made by a woman at self-delivery during a paroxysm of pain. This explanation is admissible, so long as it is confined to injuries which by any reasonable construction might be received during labour ; but if the marks have been *certainly* produced after the complete birth of the body, it will not apply.

In *R.* v. *Ancliffe* the accused was delivered of a child under much suffering, on a stone floor, and in the presence of another woman—a witness. The child was born alive, and was heard to cry several times. The witness left it in charge of its mother, and on returning shortly afterwards, she found it dead with black marks upon its throat. The midwife, who separated the child from the mother, said that it gave a sort of half-cry : she thought that it was dead when she first saw it. The marks on the neck were not more than such as a woman might have caused in attempting to deliver herself. There were many ecchymosed marks about the throat of the child as well as on the right side of the neck, and blood was effused beneath them. The marks might have been produced by the fingers ; death had been caused by pressure on the windpipe. The jury returned a verdict of " not guilty."

Isolated or detached marks of ecchymosis, as from local pressure of the fingers and thumb, cannot be set down to the navel-string. Other accidental cases may, however, come into operation. In *R.* v. *Sampson* there was a mark on the neck of the child, and it was alleged that this had been caused by pressure of the fingers, *i.e.*, by pinching the windpipe. The mark was of a red colour, and an inch and a quarter long. The medical witness stated that it was below the spot where a cap-string would usually be tied, but the mark might possibly have been caused by the knot of a tie. The accused was acquitted.

On the other hand, in homicidal strangulation, where as a rule much more violence is used than is necessary for causing death, great ecchymosis and extensive injury to the surrounding soft parts will usually be found. Sometimes all difficulty is removed by the discovery of a rope, tape, or ligature, tied tightly round the neck ; or, if this be not found, evidence that some ligature has been used will be discovered from the indentations or irregularly ecchymosed spots left on the skin—the depressed portions of skin being generally white and the raised edges livid or œdematous (*vide* " Hypostases," Vol. I).

Was the Constriction before or after Death—before or after Respiration ? A medical witness is sometimes asked whether the ligature or the fingers had been applied to the neck of a child before or after its death, or before or after it had breathed. So far as external marks of strangulation are concerned, there is no difference in the appearances, whether the constriction takes place during life, or immediately after death, while the body is warm. Marks of violence on the neck will present precisely the same features, whether the child has breathed or not, provided it is *living* and the blood circulating.

In the case of *R.* v. *Wren*, the child had breathed, and was born alive. There was a piece of tape tied around its neck very tightly, and fastened behind, and there was a discoloration of the skin beneath ; the tongue was livid and swollen, and blood was effused beneath the scalp. The medical witness admitted that the

mark on the neck might have been produced after death; and as he could not therefore positively say that the child had been strangled, the accused was acquitted. In *R.* v. *Green*, the body of the child was found with a ribbon tied around its neck so tightly that the parts on each side were swollen. Death was caused by strangulation, but it was suggested for the defence that the ribbon might have been placed there as an ornament or as part of the dress; and as the *post-mortem* examination of the body was not made until forty-eight hours after death, it was assumed that the tightening of the ligature was only apparent, and the result of a swelling of the parts after death. The accused was acquitted. In *R.* v. *Morgan* some parcel string was tied very tightly around the child's neck. The lips were swollen, the face was puffy, the tongue protruded, and there was a deep indentation around the neck in the course of the ligature. The lungs were fully distended with air, so as to leave no doubt that the child had been born living, and had been strangled.

Was the Constriction produced while Circulation was still active? The answer to this question should give a complete solution to the question, Was the child *alive* when strangled? and as a matter of fact it does so, but not to the question, was it *born* when strangled?

Inasmuch as from the first day of conception the child's circulation is always independent of the mother's, the only issue is: *If the child was strangled during or immediately after birth, medical evidence alone is quite insufficient to determine whether it was strangled before or after complete birth.*

In *R.* v. *Raven* the child was born alive, placed on the bed, and was heard crying for five to ten minutes. Fracture of the skull was the cause of death, and, according to the mother, the injury was inflicted after the child was born, but *before* the umbilical cord was severed. The jury acquitted the accused.

In another case the child was mature; the umbilical cord had not been tied; it was torn and jagged at the end. There was a bloody discharge from the mouth and nostrils. The mouth was open; the tongue protruded between the lips. There was general lividity of the head and face. On the lower part of the neck there was a well-defined circular mark or indentation about two lines in breadth. The mark corresponded to a stay-lace, with which the bundle containing the dead body of the child was tied. The skin in the indented part was thin, semi-transparent, and parchment-like. There were linear impressions of the threads of the tape to be seen on it. There was no extravasation of blood. The lungs filled the chest; they were of a bright-red colour; they weighed twelve drachms. They floated on water entire, as well as when divided into small pieces; and they floated when the divided portions were compressed. They crepitated on cutting, and when the portions were squeezed, frothy blood escaped. The cavities of the heart contained dark blood, and the whole nervous system was gorged.

In the opinion of the medical witness these facts established:—that the child was born alive; that it died from strangulation; that the mark on the neck was not produced by the navel-string, but by some ligature intentionally applied. Full and perfect respiration and an independent circulation in the child were proved. All this was admitted, but the question at the trial was whether the ligature was applied to the neck before or after the entire birth of the child. This did not admit of a positive answer, and the accused was acquitted.

It may be an important question whether, in these cases, the absence of any mark or discoloration of the skin by a ligature should be taken as evidence that the means of constriction had not been applied during life. Ecchymosis from the ligature is not a necessary consequence of constriction, either in a living or a dead child, although it might be expected

that there would be few cases of child murder or of infanticide by strangulation there would not be some ecchymosed mark or discoloration, chiefly on the assumption that great and unnecessary force is suddenly applied. Besides, a much slighter force would cause ecchymosis on the skin of a new-born infant than would be required to produce an effect on that of an adult. When there is no definite mark from a ligature, an attempt may be made by the defence to show that death could not have been caused by strangulation, as in the following case :—

The deceased child was discovered with a tape tied tightly around its neck. It was full-grown and healthy, and apparently had been born alive, as respiration had been fully established. The lungs filled the chest, floated on water, and crepitated when pressed. From the livid appearance of the face and neck, the congested state of the brain, an effusion of blood on the surface, and the ligature around the neck, the child had been strangled. The medical witnesses were not agreed whether the ligature had produced a mark of discoloration on the neck. The defence contended that the child could not have been strangled, because a tape tied so tightly around its neck as to cause death would necessarily have left a discoloration which would leave no doubt. A verdict of guilty was returned.

Was the Strangulation caused by Efforts at Self-delivery ? Cases of some interest in which effusions of blood are found beneath the muscles of the neck of new-born children occur occasionally. Such an appearance might induce a medical witness to affirm that great violence had been applied to the neck with criminal intention. They may, however, have been produced by the woman in self-delivery.

A child was found strangled with a stocking tied tightly round its throat. The woman who was charged with the murder was acquitted on the ground that she might have tied the stocking around the neck of the child in order to assist her in delivering herself. In another case, the tongue was swollen, the eyes protruded, and a tape was passed *three times* round the neck. It had been passed once around and *double-knotted*, and then passed around twice and again double-knotted on the left side of the neck. Upon removing the ligature there was a deep indentation in the neck and much discoloration. The medical witness was convinced that the child had been born alive, and had died from strangulation. He could not say whether the child was completely separated from the mother when the strangulation took place.

The defence was that the strangulation had been caused in the efforts of the mother to deliver herself when she was in all the agonies and throes of parturition, the ligature having been resorted to by her to aid the delivery. The woman was acquitted.

Juries are ready to act upon any suggestions to account for marks of violence on the neck of a new-born child.

In *R.* v. *Ashton*, the child was found dead in the soil of a privy, with a piece of ribbon tied tightly around the neck, the mark of a bruise on the head, two deep cuts in the throat, and about seventeen punctured wounds on the body, one of which had penetrated the heart. The medical witness stated he had no doubt the child was born alive, and that these injuries were the cause of death. The defence suggested that there was no proof of existence after entire birth of the body, and that the injuries found were "very probably the result of accident in the course of self-delivery by an unhappy young creature like the prisoner." The jury accepted this inconsistent view of the medical facts, and acquitted her. In *R.* v. *Parkinson*, some suspicious marks on the neck of a child were referred to the accidental tightening of the string of a cap.

In order to prove in such cases that the ligature was placed around the neck from homicidal motives there must be a careful examination of

the material, the position, and the nature and number of the knots, any one of which may possibly constitute a positive piece of evidence. If the material used were kept in a drawer in a distant room and the knots had been tied with great care under the chin (in itself suggestive of homicidal violence), would any medical witness swear that it was impossible for the mother while in labour to walk to the distant room and to tie the ligature around the presenting head and neck ? For other remarks *vide* Vol. I, pp. 565 *et seq.*, where homicidal strangulation is dealt with.

Accidental Marks resembling those of Strangulation. On the fore part of the neck of a child a mark or depression is sometimes accidentally produced by forcibly bending the head forwards on the chest, especially when this has been done repeatedly and recently after death, while the body is warm. It may occur, also, as an accident during labour. Such a mark must not be mistaken for the effect of homicidal violence.

Marks simulating violence are sometimes found on the necks of new-born children :

A child was expelled rather suddenly ; and after making two or three convulsive gasps, it died. Whilst endeavouring to restore animation, the accoucheur observed a bright-red mark extending completely across the upper and fore part of the neck, from one angle of the lower jaw to the other, as though it had been produced by strangulation with a cord, except that the mark was not continued round to the back of the neck. It was of a vivid red colour, and not like a bruise or ecchymosis : it had very much the appearance of a recent excoriation. It was most clearly defined in front, where it was about a quarter of an inch in breadth, and it became diffused at the sides. The face was not swollen, and there was no fullness of the veins.

A distinction in this case might have been based upon the colour of the mark, unabraded state of the cuticle, and the absence of congestion of the face and venous system. Nevertheless, the case is of importance, and the facts should be borne in mind. In another case there were red marks on each side of the nose of a new-born child which were mistaken for the effects of violence applied to the nostrils during an alleged attempt at suffocation. The marks were examined closely, and it was concluded that they were nævi and had nothing to do with the death of the infant.

Littlejohn[1] describes and illustrates a case in which a furrow on the neck of a child closely resembled a constriction mark.

The subject of strangulation may be summarised as follows :

Congestion of the face and head in a newly born child is not a proof of death from strangulation.

A child may be strangled during birth by the accidental twisting of the navel-string around its neck.

The navel-string, like any other ligature, may produce a livid or ecchymosed depression on the neck.

The marks on the neck, arising from accidental causes, may resemble those which arise from strangulation.

The local effect of constriction on the neck, either by the navel-string or any other ligature, is the same if the child be *living*, whether it has or has not breathed.

The effect is the same whether the child has been *partially* or *entirely* born.

[1] " Forensic Medicine," 1925, p. 82.

The effect of a ligature on the neck of a *living* child is the same whether the navel-string has or has not been severed.

A newly born child may die from strangulation, without the fact being necessarily indicated by ecchymosis on the neck. This depends on the nature of the ligature, and the amount of force used.

Cold and Exposure

A newly born child may be easily destroyed by exposing it uncovered, or but slightly covered, to a cold atmosphere. In a case of this kind there may be no marks of violence on the body, or these may be slight and evidently of accidental origin. In death from cold the only appearance occasionally met with has been congestion of the brain, with or without serous effusion in the ventricles. (See "Cold," Vol. I, pp. 480 *et seq.*). The evidence in these cases must be purely circumstantial. The medical witness may have to consider how far the situation in which the body was found, the kind of exposure, and the temperature of the air, would suffice to account for death from the alleged cause. A newly born child is easily affected by a low temperature, and warm clothing is required for the preservation of its life. An inspection of the body should never be omitted on these occasions, because there may be some cause of natural death which would at once dispose of any criminal charge. Assuming that the child died from cold, it becomes necessary to inquire whether the mother exposed it with homicidal intent. As a rule a woman recently delivered does not expose her child for the purpose of destroying it, but for the purpose of abandoning it; hence it is rare to hear of convictions for child murder or of infanticide where cold was the cause of death.

Starvation

A newly-born child kept long without food will die, and no evidence of the fact may be derivable from an examination of the body. There may be no marks of violence externally, nor any pathological changes internally, to account for death. This is a rare form of murder or of infanticide, except that it may be incidentally combined with exposure or abandonment. The only appearance likely to be found on examination of the body would be complete emptiness of the alimentary canal. Without corroborative circumstantial evidence this would not suffice to establish the cause of death: a medical witness could only form a probable conjecture on the point. In a suspected case of this kind, the contents of the stomach should be tested for farinaceous and other kinds of food (*vide* "Live Birth," *ante*).

Violence by Wounds (External and Internal)

The features which have been already described in Vol. I, pp. 259 *et seq.*, as peculiar to wounds, contusions, and fractures inflicted during life, may be met with in a child whether it has breathed or died without breathing. These features are open to the exceptions there pointed out; for they will also be present, assuming that the wounds were inflicted immediately after the cessation of respiration or circulation in the child, or after the cessation of circulation only, if the act of breathing has not been performed.

When a child is murdered it is usually found that the amount of violence inflicted is considerably greater than that which is required to destroy the life of a newly born child, and in this way satisfactory proof of the crime is usually obtained.

The questions that the medical witness will have to answer in such cases are :—

1. Were the wounds inflicted before or after birth ?
2. Were the wounds inflicted before or after death ?
3. If before, did they cause death ?
4. Were they accidental or homicidal ?

1. **Were they inflicted before or after Birth ?** In most cases it will be impossible for a medical witness to return any answer to a question put in this form. Medical evidence may show whether a child was living or not when the wounds were produced, but not whether the *whole* of its body was or was not in the world at this time. In a few cases an opinion may be formed from the nature, extent, and situation of these injuries.

The question whether the injuries were inflicted before or after birth is really the whole crux in cases of alleged child murder or of infanticide.

There are, however, a number of injuries in the wider sense that are commonly recognised, or recognisable by a little consideration, as ante-natal.

A practitioner must remember that if, while in an advanced state of pregnancy, a female should **accidentally fall** the child may sustain an injury by a blow through the abdominal walls, and the fact is of sufficient importance to merit attention, as the following case will show :—

A pregnant woman, within five days of the ordinary term of gestation, fell while running, and her abdomen struck against an angular stone. There was an immediate loss of blood, and the movements of the child ceased. Parturition commenced four days after the accident. The head of the child much enlarged, and in a putrid state. The woman died within an hour. Upon examining the child, the skull was found almost crushed, the parietal having become separated from the temporal bones as if by external violence. The marks of injury were entirely confined to the head.

In injuries of this kind resulting from falls it is probable that the child will be born dead ; there may also be marks of violence on the abdomen of the woman. Many observers have described cases in which the limbs of the fœtus *in utero* have become deeply indented or spontaneously amputated by the twisting of the umbilical cord around them (*vide ante*, p. 175) ; and this is now generally regarded as the cause of ante-natal amputations. Such accidental injuries before birth could not possibly be mistaken for violence inflicted on the body of a child after its birth.

A child may be accidentally destroyed in the act of birth by the **neck being forcibly twisted**, whereby a displacement of the cervical vertebræ, with injury to the spinal marrow, may occur and cause death. Such injuries are immediately discovered by an examination of the body. The neck of a child is very short, and it possesses considerable mobility.

Non-professional persons may, when a woman has been secretly delivered, ascribe the tumours known as **Caput succedaneum and Cephal-hæmatoma** to violence, whereas they are in fact produced by natural causes. The swelling is generally situated on one of the parietal bones, its situation depending on that part of the body which presents during

delivery. After the discharge of the waters, the scalp is firmly compressed by the mouth of the womb. This pressure interferes with the circulation through the skin, and causes the compressed portion of the scalp to swell. In the simplest form of this tumour serum only is found in the swollen part; occasionally this is mixed with blood, and there are small ecchymoses beneath or in the scalp and pericranium, or even within the skull, but there is generally no injury to the bones, nor is there any laceration of the skin externally. In other cases blood in some quantity is found effused in the tumour either under the scalp, the membrane covering the skull (pericranium), or within the skull itself.

Violence from blows or falls which would produce bloody effusions beneath the scalp, or within the skull, would in general be indicated by injury to the skin or by fracture of the bones. The following case shows that caution is required in forming an opinion.

A child died twenty-three days after birth. The tumour (cephalhæmatoma) was about the size of a walnut originally, but it had extended so as nearly to cover the right parietal bone. On dissection it was found to be filled with coagulated blood, beneath which was a layer of dense fibrinous matter. The right parietal bone presented a fissure with clean edges running from the coronal suture obliquely backwards and upwards. On the inner surface of the bone was an effusion of blood between the cranium and dura mater more than half an inch in thickness, and occupying the whole of the hollow of the parietal bone. There was no reason to doubt that the fracture and effusion were the results of compression during delivery; they had not been occasioned by external violence.[1]

Vide also cephalhæmatoma in works on the surgical diseases of children or text-books of midwifery.

Of course if a fracture is present (*vide* below) such a hæmorrhage is to be expected, but quite apart from this a **meningeal or even an intracerebral hæmorrhage** is a well-recognised consequence of difficult labour, and a well-recognised cause of death during or shortly after birth. When such is found, then, in a dead child, it must not be hastily assumed that it necessarily indicates violence; all other factors must be taken into consideration—length of labour, other marks suggestive of violence, etc.

2. **Were they inflicted before or after Death?** Although marks of violence may establish that a child was living at the time they were inflicted, they can never show that it was *born* alive. Injuries met with on the bodies of children alleged to have been born dead ought, however, to be of such a nature as to be readily explicable on the supposition of their having arisen from accident. If, from their nature, extent, or situation, they are such as to evince a wilful design to injure, it is a fair ground for a jury, and not for a medical witness, to inquire why these extensive wounds, or other marks of violence, were inflicted on a child, if, as it is alleged, it was really born dead. It must be confessed that in such a case there would be a strong moral presumption of murder, although medical proof of life or of live birth might totally fail.

As a summary of these remarks, it may be observed that, although physiologically a child may live for a certain period after its birth without breathing, and legally its destruction during this period would amount to murder, yet there are at present no satisfactory medical data to enable a witness to express a positive opinion on this point. If other evidence

[1] "Trans. of Med.-Chir. Soc.," vol. 28.

were adduced of a child having lived and having been destroyed in these circumstances—as where, for example, a woman causes herself to be delivered in a bath of water, or an accomplice covers the mouth of an infant in the act of birth, or immediately after it is born—a medical witness would be justified in asserting that the absence of the signs of respiration in the lungs was no proof that the child had been born dead. Indeed it is apparent that the process could not be established, owing to the criminal means actually employed to prevent it. In general, those cases in which questions relative to life before respiration might arise are stopped in the coroner's court—the usual practice being, when the signs of respiration are absent or imperfect, to pronounce that the child was born *dead*. If the lungs sank in water, the presence of marks of violence on the body would be considered as furnishing no evidence; for the sinking of the lungs would be taken as positive evidence of still birth, an incorrect inference (*vide* pp. 144 *et seq.*, the hydrostatic test).

The following case was the subject of a criminal charge at Havre:

A woman was delivered of twins. As soon as the first child was born, but not before it had breathed, she killed it by fracturing its skull with a wooden shoe. In a few minutes afterwards the second child was born; but scarcely had its head presented when she seized it and fractured its skull in a similar manner. On an examination of the bodies of both children, the same degree of violence was found, presenting in each case precisely similar features. From the appearance of the injuries, they must have been inflicted on both children at a time when the circulation was going on. In one child, however, it was proved that respiration had taken place; in the other that it had not. In the latter case many practitioners would at once have affirmed that the child had not lived, because there was no proof that it had respired; and they would have proceeded to draw the inference that this could not have been a case of infanticide. Bellot, however, stated that, although the child had not breathed, he had no doubt it had been *born alive*, and that it would have lived to breathe but for the violence inflicted.

Apart from this ever-recurring question about breathing, it is as easy in a child as in an adult to determine whether an injury was inflicted before or after death, and the question is decided on precisely similar lines (*vide* Vol. I, pp. 299 *et seq.*).

3. **If before Death, were they the Cause of Death?** This point must be considered on precisely the same lines as in the case of wounds found on an adult (Vol. I, pp. 264 *et seq.*), but it must be remembered in drawing final conclusions that a frail infant may succumb to injuries which are comparatively slight. Newly born children bear a loss of blood very badly.

In *R.* v. *Wood*, the main question was, whether five severe wounds found on the head of a child were inflicted before or after death, and accidentally or criminally. The mother confessed that the child was born alive and had cried, but asserted that it had died in five minutes after its birth. It was assumed that the wounds might have been accidentally inflicted after death with a spade which had been used for the burial.

The medical witness attributed death to the wounds, which, in his opinion, could not have been accidentally produced; but he admitted, in cross-examination, that the wounds would have presented the same appearance if they had been inflicted immediately after death. Apparently no microscopic sections had been made, otherwise the witness might

possibly have been able to prove that the wounds had been inflicted before death. Answers to questions of this kind can be given most positively where the body has been examined soon after the infliction of the wounds. It is dangerous to pronounce an opinion when the child had been long dead.

4. **Were they the Result of Labour ? Were they accidental or homicidal ?** The answer to this question must depend upon the nature and the situation of the injuries. To exhaust the possibilities under either heading would require a complete monograph with very numerous illustrative cases, for in each case it is necessary that in both respects the tale of the accused should be compared point for point with the details of the wounds, and each of these may vary infinitely in details.

Evidence from the Nature of the Wound. Nothing in the shape of a punctured wound, or of a clean incised wound, or of a burn, etc., could occur as the result of a natural labour. Whereas occasionally uterine force might produce dislocation of a joint or even fracture of a limb, it is well known and admitted that a caput succedaneum and cephalhæmatomata are usually produced by protracted labour, and that fractures of the skull (*vide* below) are occasionally caused in this way. It must be noted that we are here speaking of actual wounds, not of causes of death in protracted delivery (*vide* p. 168).

Incised wounds found on the body of a new-born child may be referred to the use of a knife or scissors in attempting to sever the navel-string ; and they may therefore be due to accident. This point should not be forgotten, for a wound even of a severe kind might be thus accidentally inflicted. In such cases we should always expect to find the navel-string *cut*, and not lacerated. The end of it should, for the purpose of examination into this point, be stretched out on a piece of white card.

In *R.* v. *Wales*, there was a wound on the right side of the neck of the child, not involving any important blood vessels, although it had caused death. The medical witness admitted that it might have been accidentally inflicted. The accused was acquitted.

As a similar question may be unexpectedly put at a trial, a witness should prepare himself for it by making a careful examination of the wound and of the navel-string. This will usually suffice to show whether an incised wound has been produced accidentally in the manner alleged, or with homicidal intent.

In *R.* v. *Hacking* there could be no doubt from the nature of the wound that it had been inflicted with homicidal intent.

A woman was charged with the murder of her infant child. The medical evidence was that its throat had been cut with some thin-bladed sharp instrument — *a portion of the gullet and windpipe having been cut away*. The woman stated that the child was born dead, and confessed that she had, as she believed, cut its throat with a penknife, which she had afterwards wiped and put away. The weapon was found in her pocket. The medical witness stated that the child had certainly *breathed*, and he was inclined to think that it had been *born alive*. He admitted that a child might breathe when partially born, and die before it was wholly born ; also that the appearance of the wound, whether inflicted before or immediately after death, would be similar ; and that it was impossible, from the examination,

to say whether the child had been partially or wholly born at the time of its infliction. The woman contended that there was no evidence that the child had been *fully born alive*. The woman was acquitted.

Such a wound with a penknife was hardly likely to be inflicted on the child by any accident, or for the purpose of aiding delivery. As the child had breathed, it is absurd to suggest that the woman waited until it had died from some other cause, as to which there was no sign; and that after death, without any conceivable motive, she had cut out a portion of its throat. The acquittal appears to have been based on the assumption that the child was destroyed before it was wholly born; and although it had breathed, there was no evidence to show that breathing had continued after its body was entirely in the world.

Small punctured wounds of vital organs, brain, cord, heart, etc., should be most especially looked for by a most thorough autopsy.

The spinal marrow is said to have been wounded by needles or stilettos introduced between the vertebræ, the skin having been drawn down before the wound was inflicted, in order to give to it a valvular character, and to render it seemingly superficial. The brain is also said to have been wounded, by similar weapons, through the nose or the thinner parts of the skull (the fontanelles).

Fractures of the Skull. These may arise from uterine action, accident by fall, or criminal violence, and may be equally met with in violence to the living or recently dead body.

Although it has been a matter of frequent observation that great violence may be done to the head of a child during parturition without necessarily giving rise to fracture, yet it is beyond all doubt that such an injury may occur by the expulsive efforts of the womb forcing the head of a child against the bones of the pelvis. Even the violent compression which the head sometimes experiences in passing the mouth of the uterus may suffice for the production of fracture.

Fractures of the skull are uncommon accidents of parturition. They usually result from the pressure of the sacral promontory, and are seen as a spoon-shaped or gutter-like depression. Fissured fractures may also be caused in natural delivery, especially in contracted pelves, but they are usually the result of delivering the head with forceps.

These accidental fractures are generally slight: they commonly amount merely to fissures in the bones, beginning at the sutures, and extending downwards for about an inch or less into the body of the bone. The frontal and parietal bones are the only bones likely to be fissured or fractured during the act of parturition. In the greater number of cases reported, the parietal bones only have presented marks of fracture.

Fractures are always associated with hæmorrhage within the skull, but intracranial hæmorrhage without fracture is much more common in causing the death of the child during birth.

In one instance a woman was delivered after a labour of twenty-seven hours. While the head of the child was at the outlet, the uterine contractions ceased for an hour; the child was then suddenly expelled, and Schwoerer received it in his hand, so that its body did not come in contact with anything that could produce physical injury. The child did not breathe when born, but it showed evident

signs of life. The pulsations of the heart and umbilical cord were distinctly perceived; these gradually ceased, and no effort could restore the child or bring about respiration.

The most important fact observed was the condition of the head. There was a considerable swelling of the skin at the top of the head, chiefly over the right parietal bone, and beneath this a quantity of dark-coloured blood was effused. Two fissures or slight fractures were perceived in this bone—one passing from the sagittal suture towards the centre of the bone, about half an inch in length; and a second, about an inch long, passing from the lambdoidal suture at the back part of the parietal bone, also towards the centre. There was no doubt that these fissures or fractures in the bone, with the effusion of blood beneath, were produced by the action of the uterus alone during delivery.

From these appearances, and in an absence of all knowledge of the facts, the following conclusions might well have been drawn: first, that this child was born capable of living, and probably lived after its birth; and second, that it had died a violent death from injuries inflicted on the head. A woman delivered of an illegitimate child in secret might, although innocent, have thus been compromised in a charge of murder. As the lungs sank in water, entire and divided, it is highly probable that the case would have been stopped by a coroner's jury on medical evidence that the child was stillborn. If, however, further proceedings had been taken, the amount of violence to the head was too slight to justify a medical opinion that it conclusively indicated an act of murder. The bones were merely fissured, not dashed in or displaced, and the brain was uninjured; the fissures were slight, and the amount of blood effused was very small for an act of homicidal violence involving the skull. Medical men must be prepared to make full allowance for the accidental occurrence during labour of such injuries as these.

In the following case it is probable that a fracture of the head of a child was produced by the expulsive action of the uterus.

The body had been found secretly buried; it was fully developed, but the child had evidently not breathed. The navel-string had been cut and tied; but six inches of it still remained attached to the body. On the left side of the cranium, near the summit, there was a small effusion of blood; below this, a fissure half an inch in length was found in the edge of the left parietal bone, close to the line of the sagittal suture, and near the posterior fontanelle. On shaving off the hair there was no discoloration, nor any mark on the skin indicative of a blow. There was no evidence to show that any violence had been used to the child at its birth, and from the description of the fissure it was a fair presumption that it had arisen from the muscular contractions of the uterus during delivery.

The possible occurrence of an accidental injury of this kind has been strained, in several cases of child murder or infanticide, to explain the origin of fractures which, however, could not be fairly assigned to such cause.

In *R.* v. *Irwin*, there was no doubt, from the state of the lungs, that the child had fully breathed, and there was violence to the head which satisfactorily accounted for its death. The whole extent of the right side of the head was deeply bruised, and there was a large quantity of coagulated blood lying beneath the bruise. In the centre of the right parietal bone there was a fracture extending across the vertex for fully four inches, and involving a part of the parietal bone on the opposite side; it was in a continuous even line, not radiated and not depressed. The pericranium, bones, and soft parts in the track of the fracture were deeply ecchymosed, while on the surface of the brain, particularly on the right side, there was a copious effusion of clotted blood. It was impossible to refer severe injuries of this kind to the action of the uterus in delivery, or to violence applied after death. The accused alleged that the child was stillborn.

In the case of *R.* v. *Mussett*, the head of a child was almost flattened from the violence sustained. It was clear that no fall or other accident could explain this condition. Some fresh blood and a single hair were found on a shelf in the cellar, for which the accused accounted by stating that she had killed a rabbit there.

A microscopical examination, however, showed that it was human hair, and not the hair of a rabbit. The medical evidence established from the state of the lungs that the child had breathed, and that it had had an independent existence. The accused was convicted.

The following case of spontaneous fracture of the left parietal bone occurred during a natural but tedious labour, in which the head of a child was five hours in the pelvic cavity, although the pelvis was well formed.

There were three fissures in the bone—one running into the sagittal suture, one to the inner inferior angle, and the other to the middle of the anterior edge of the bone. The child was stillborn. Much blood was effused beneath the scalp, but there was none under the skull.

These accidental fractures and effusions of blood from uterine action are usually recognised by their very slight extent. In cases of murder by violence to the head, the injuries are usually much more severe; the bones are driven in, the brain protrudes, and the scalp is extensively lacerated. Such severe injuries as these cannot arise accidentally from the action of the uterus during parturition.

A case occurred in which, in addition to severe injuries to the brain, coal-dust and minute pebbles were found driven into the skin of the head as a result of the body being thrown from a height.

It may sometimes be urged that the woman was unexpectedly seized with labour, that the child was expelled suddenly by the violent contractions of the uterus, and that the injuries might have arisen from its head coming in contact with some hard surface—as a floor or pavement. A woman may be thus suddenly and unexpectedly delivered while in the erect posture, although this is not common among primiparous women, and severe injuries may be thus accidentally produced on the head of a child.

In a case of sudden delivery in the erect posture in a primiparous woman, without injury to the child, the umbilical cord was ruptured at the distance of about two inches from the navel. In another case, a woman who had borne a child was suddenly delivered while standing. The child fell to the floor on its vertex, and the cord was ruptured. A small quantity of blood escaped from the part struck, there was no fracture of the bones, and the child sustained no serious injury. In the case of another primiparous woman, sudden delivery took place while she was in the act of sitting down. The child was forcibly expelled, and fell with its head on to the floor of the room; it was taken up dead, the cord being still attached to it, and the placenta, which came away shortly after the birth of the child.

Fractures of the skull even in these circumstances are of rare occurrence. Out of 183 cases reported by Klein, in which the women were rapidly delivered while sitting, standing, or inclined on the knees—the child falling on the ground or floor—there was only one instance in which a child was killed; and there was not a single instance in which the bones of the skull were fissured or fractured so far as could be ascertained by external examination. Chaussier performed some experiments on the bodies of stillborn children, allowing them to fall with their heads downwards on a paved floor, from a height of eighteen

inches ; and he found that one or other of the parietal bones was fractured in twelve out of fifteen cases. Although these results are conflicting, yet Klein's observations appear more to the purpose, because they were made in circumstances in which the question would really arise in a case of infanticide. They are, moreover, strikingly supported by numerous cases reported in the medical journals of children being delivered while the mother was standing up, falling head first on to various hard surfaces and yet only receiving bumps and bruises without fracture.

These reported cases lead to the inference that although fractures of the skull are not likely to occur, the *possibility* of their occurrence cannot be denied.

A fracture of the skull of a child may be produced when a woman is delivered in the erect position. In a reported case there was merely the appearance of a bruise on the head, and the cord was ruptured (not cut) three inches from the navel. The child did not suffer from the fall, and continued well until six days after its birth, when it was seized with convulsions and died. There was a fissure of about an inch and a half in length in the upper part of the left parietal bone. A clot of blood was found in this situation between the dura mater and bone, and there was congestion of the vessels of the membranes ; with this exception there was no morbid appearance of the body. In another case in which the child died from injury to the head by falling from the body of the mother in an unexpected delivery, the cause of death was effusion of blood on the brain. There was sudden delivery in the erect posture, the child falling with its head on a deal floor. A large fissure was found in the right parietal bone, and there was a great effusion of blood, which had caused death. In this, as in some other cases of delivery in the erect posture, the umbilical cord was torn through about two and a half inches from the body.

In a similar case the mother was charged with the murder of her child. The right parietal bone was fractured, and there was effusion of blood internally, but there was no mark of external violence. The cord had been ruptured at a distance of two and a half inches from the navel. The stomach of the child contained the usual albuminous and mucous matters of the fœtal state, without any appearance of food. The lungs were inflated and highly crepitant ; the foramen ovale and the ductus arteriosus were in their fœtal condition. The child had probably been drowned in the discharges through lack of assistance at the time of birth. The woman, who admitted that the child had fallen from her suddenly, was acquitted. In each of four cases of sudden delivery the child dropped from the woman, and in two of them there were fissures in the parietal bones. The children recovered from the effects of the accidents. Other cases of rapid delivery in the erect posture are reported. In these there was no injury to the child, although in one case the delivery took place on the deck of a vessel.

Braxton Hicks called attention to the possibility of fractures or fissures of the bones of the head being caused by lateral pressure of the skull from the hands of the mother in order to aid her delivery. It would appear, however, from his experiments on this subject that such injuries may, as a rule, be distinguished from those which are the result of a deliberate attempt at murder.

Hicks performed various experiments on the heads of still-born infants.

In one instance by sudden lateral pressure he produced a fracture through the arch of the cranium, but the bones generally yielded to the force without breaking. When, however, one side of the head was laid on a hard and resisting surface like the floor, and the other side was compressed firmly and suddenly, a fracture was produced in the lower parietal bone, although the bones of the head were firmly ossified. In two other experiments on large children with firmly

ossified skulls, lateral pressure with both hands, one on each side, caused no fracture or injury such as could be mistaken for homicidal violence. There was a fissure of about half an inch in the left parietal bone, produced not so much by pressure as by an indentation of the bone. The appearances produced by pressure on the head of a stillborn child, after a severe labour, were as follows :— There was a large bloody scalp-tumour over the right parietal and occipital bones ; liquid blood oozed out on section ; and the veins on the inside of the skull were highly congested, especially on the part beneath the scalp-tumour.

The fissure produced on the parietal bone was, however, too slight to be consistent with the theory of homicidal violence. It follows, from these experiments on the dead bodies of newlyborn children, that fractures of the skull are not easily produced in the conditions in which a woman would be placed in delivering herself. The bones, as in natural delivery, yield to great pressure without breaking. Their composition and elasticity, as well as the yielding of the parts, in the situation of the sutures, tend to counteract the effects of manual violence thus applied to the head.

When the marks of violence found on the head, neck, or body of a child cannot be easily referred to an accidental fall, it is common to ascribe them to the **efforts made by a woman in her attempts to deliver herself,** the destruction of the child being an accidental result of these efforts. A medical opinion in such cases must depend upon the nature, situation, and extent of the injuries ; and each must be therefore decided by the circumstances attending it.

A medical witness will often find himself questioned respecting the strength or capability for exertion possessed by working-class women shortly after childbirth. Many medical practitioners, judging only from what they have observed among the middle or higher classes, are liable to be led into an erroneous opinion, which may affect their evidence.

A woman accused of child murder walked a distance of twenty-eight miles in a single day, with her child on her back, two or three days after her delivery. Cases have been reported in which women have walked six and eight miles, on the very day of their delivery, without sensible inconvenience. In one case the woman was engaged in reaping—she retired to a little distance, effected her delivery by herself, and went on with her work for the remainder of the day, appearing only a little paler and thinner than usual. In the case of Macdougal, the accused, who was sleeping in bed with two servants, arose, was delivered, and returned to bed without either of them being aware of what had occurred. Cases like the last have often occurred.

A firm resolution, with a strong desire to conceal her shame, may enable a woman to perform, immediately after her delivery, acts connected with the disposal of the body of her child, which, from ordinary experience, might appear to be much beyond her strength.

In *R.* v. *May*, a domestic servant had been sent to market with some poultry. On her return, she asked the boy who drove the cart to stop. She got out and went to a recess by the side of the road. In five minutes she was observed following the cart, and she walked home a distance of a mile and a half. She went about her usual work on that and the following day. She had been delivered of a child in the recess, and it was subsequently found there. One witness heard it cry, but it soon died.

Vide also p. 47 for further references.

A witness, however, should be prepared to admit that a woman at the time of her delivery, owing to pain and anxiety, may be deprived of all judgment, and may destroy her offspring without being conscious of what she is doing. (See the Infanticide Act, 1938.) Mere appearances of violence on a child's body are not *per se* sufficient, unless there is some evidence to show that the violence was knowingly and intentionally inflicted, or the appearances are of such a kind as of themselves to indicate homicidal intent.

When the skull of a new-born infant is found to be fractured, the question put to a medical witness may be—Is the degree of injury such as to be consistent with the view that it was accidentally caused during delivery, either by the woman herself, or by some person who was present ? Braxton Hicks was called by a midwife to aid the delivery of a woman. On examination, he observed as follows :—

The skull was fractured through the parietal bone on one side, and there was a slight fracture of the edge of the occipital bone, with a scalp-tumour. The head of the child was at the brim of the pelvis, and the fractures had been produced by the midwife in her attempts to push the head back into the cavity. The woman was delivered by instruments : in such a case a woman would not be able to deliver herself. In another instance a new-born child had a fracture through the arch of the skull, from one side to the other, and a fracture in the frontal bone on one side. The jaw was broken, the angle of the mouth lacerated, and the humerus was also fractured. The woman who had been delivered of the child was charged before a magistrate with wilful murder ; and the question was, had she, in attempts to aid delivery, produced this violence on the body by seizing the head and violently compressing it, or had the injuries resulted from the body falling on the floor of the room ? Conflicting medical opinions were given, but Hicks, who was called as an expert, admitted that the injuries might have been possibly inflicted by the woman on the child in her attempts at self-delivery.

Evidence from the Locality of the Wound or Wounds. If many incised wounds are found about the limbs or body of a child, infliction by a second person, and that not by accident, is an inevitable deduction. In some instances the body of a child is found cut to pieces, and the allegation in defence may be that the child was stillborn, and the body had been thus treated for the purpose of concealment.

Toulmouche has reported a case of this kind. As the woman had not destroyed the lungs, experiments on these organs gave satisfactory evidence of complete respiration. The cavities of the heart and great vessels were empty : the body was generally drained of blood, and the skin throughout very pale. This led to the inference that the mutilations must have been inflicted while the child was living ; and as all the parts were healthy, and no natural cause of death was apparent, Toulmouche ascribed the death of the child to the wounds. The woman was convicted.

If marks of violence, apparently inflicted about the same time, are found on **different and remote parts of the body,** and these marks bear the characters of those produced during life, it is rendered probable that they were inflicted criminally upon a child that was completely born. Marks of severe violence on one part, as the head or breech, would not always justify such a presumption, because it might be fairly objected that they had been unintentionally produced by the woman in her attempts at self-delivery. It would be for a witness to form an opinion from the circumstances accompanying the particular case, whether they had been thus occasioned.

A child which was said to have been born dead was exhumed two days after burial and eleven days after birth. It was full grown, not putrefied, and the skin was pale and free from lividity. There was a clean cut on the right arm, dividing the fascia and muscles, as if made by a sharp instrument. The edges were much retracted, and the whole of the wound was of a florid red colour; but there was no swelling nor appearance of inflammation. There was a large vesicle (like the blister of a burn) on the scrotum, containing three drachms of a yellow-coloured serum. On the right leg, the muscles were exposed for nearly the whole length: the surface of the wound was of deep scarlet colour, and the margin widely inflamed. It had the appearance as if fire had been applied to the leg, although there was no sign of charring. These facts tended to show that the child was living when the injuries were inflicted; while the nature and situation of the injuries rendered it impossible that they could have arisen from any accident during delivery. The state of the lungs was somewhat remarkable: the *left* floated freely on water, and there was distinct crepitation in it; the right sank in water, no portion of it, when divided, was observed to float. From the buoyant and crepitant state of the left lung, there was reason to presume that if respiration had commenced during birth, it had continued afterwards. It was inferred that the child had been born alive; and that inference was corroborated by the marks of violence. It is probable that the child did not live long after birth.

The air could not have been derived from putrefaction nor artificial inflation: therefore the only question was, whether the child had breathed after its body was wholly in the world. The facts above mentioned justified the inference drawn. From a confession subsequently made by the mother, it appeared that the child had been born alive, and had cried, but, owing to the injuries inflicted on it, it did not survive birth longer than a quarter of an hour.

The **head and neck** are injured most frequently both in nature's efforts at expulsion and in a woman's frantic efforts at self-help. It is consequently in this locality that we meet with the greatest difficulty, firstly in deciding whether the injury was inflicted by nature or accident without the intervention of a second person, and secondly in deciding whether the obvious intervention of the woman was criminal or otherwise.

A medical witness would find no difficulty in determining the probability of an explanation of the origin of fractures of the neck and head, etc., if he were made acquainted with all the facts connected with the delivery. But it will, usually, be out of his power to obtain this knowledge. Sometimes the fractures will be accompanied by incisions, punctures, or lacerations of the scalp or face: in such cases, although the origin of the fractures might be accounted for by an alleged fall during parturition, the cause of the other injuries would still remain to be explained. Injuries of this nature, with the fact that there are bruises or contusions as well as fractures not connected with each other in various parts of the skull, and depending on different acts of violence, would be inexplicable on the hypothesis of an accidental fall.

A girl was delivered in secret. She at first denied that she had had a child, but afterwards produced the dead body. It was mature and had breathed. There were some marks as of pressure about the neck, and extensive effusions of blood beneath the scalp in various parts of the head. There was no gross fracture, but a fissure in one of the bones of the head. She said she had been suddenly delivered while standing up, and found that the child had fallen from her and was dead. Casper considered that this would not explain the condition of the dead body, which presented the effects, not of one, but of several distinct acts of violence.

and the death of the child was referred to the injuries found on the head. The girl afterwards confessed that she was delivered while lying on the bed, and that she had then struck the child on the head and body with a wooden shoe.

An inquest was held on the body of a female infant who had been born, according to the statement of the mother, in the pan of a water-closet on the ground-floor of the house, and was afterwards carried by her up two flights of stairs, and placed beside her in bed. She admitted that the child had been born alive, but stated that it was dead when she lifted it up from the pan to carry it to the bedroom. The navel-string was torn at a distance of four inches from the abdomen. The child, she alleged, had fallen into the water-closet pan. No trace, however, of blood or other discharge was found on or near the seat of the closet; while upon the opposite side of the closet the floor was stained with blood, which had been imperfectly wiped up. The body was that of a well-formed mature child, weighing seven pounds. The lungs had been fully expanded, covered the heart, and floated on water with and without the latter organ. The scalp presented no trace of injury; there was only the usual scalp-tumour, and on dividing the skin there was some ecchymosis at this part. The bones of the skull were extensively fractured. There was a horizontal fracture nearly an inch long over each orbital prominence; and upon the right frontal eminence the bone was broken and depressed, in an acute triangular form, three-quarters of an inch in length. The parietal bones on each side were fractured vertically from their eminences downwards, to the extent of an inch and a quarter; and on the left side the lower end of this fissure was joined by another of similar extent, passing horizontally forwards at the right angle to the edge of the bone. Similar smaller fractures were found at different parts of the upper surface of the skull, apparently not connected with each other. Within the cranium, blood was extravasated on the surface of the brain, and in the membranes. No fractures were detected at the base of the skull. It was very doubtful whether the body of the child had been in the pan at all. Assuming that the child had thus fallen, the distance was too small to have caused such an amount of injury, found on several parts of the skull; also, as the child would have passed in an oblique direction forwards from the outlet, it would probably have glided safely down by the side of the pan. In the absence of evidence as to the mode in which the injuries were inflicted, it was suggested that they might have been caused by the mother having fallen upon the child on her way upstairs; and this hypothesis was ultimately adopted by the coroner's jury, though there was no doubt that the child's death was caused by the injuries to the head.

In *R.* v. *Gibson* the skull of the child was fractured, and much blood was effused on the brain. The right lung contained air, and the left lung also, but in small quantity: they both floated in water. The accused admitted that the child cried twice and she accounted for the fracture of the skull by asserting that the child had dropped from her in a lane. She wrapped it up, and soon afterwards found that it was dead. A stone having blood and hair upon it was picked up near the body. The accused was acquitted.

In *R.* v. *Strangeways* there was not only a fracture of the right parietal bone, but the throat was cut with a knife. From the state of the lungs it was evident that the child had breathed, but the medical witness declined to say that it had had an existence independently of the mother. The defence was, that the child had dropped from her while she was standing at her work, and that it fell on the kitchen fender. There were, however, no marks of blood on the fender, and the wound in the throat was inconsistent with such a statement.

Severe fractures with great depression of the bones, and the co-existence of lacerated **wounds of the scalp with severe injuries on other parts of the body**, are not consistent with the theory of their production in self-delivery. Nevertheless, as in the following case, such violence even when plainly homicidal may be treated as accidental or as justifying a verdict of infanticide.

The medical evidence showed that the newly born child had breathed, and there was no apparent natural cause for death. There were marks of finger-nails in the neck, *evidently indicating attempted strangulation*. The bones on each

side of the head were crushed *inwards*; there was much blood effused between the dura mater and the skull, and this had caused pressure on the brain. A fall from a standing labour, or accidental force applied during delivery, could not have produced these appearances. They were caused, in the opinion of two medical witnesses, by the direct application of violence to the head of the child, and more than one blow must have been given to have produced them.

In charging the jury upon the cause of death, the judge said that the medical men had attributed it to the combined effect of strangulation and violence to the head. It might, however, be a theory that the struggles of a young woman during parturition might have occasioned the injuries, or she might in her weakness have fallen upon the child while on the floor. Did the medical evidence satisfy them beyond a reasonable doubt that this young woman had murdered her child? The jury returned a verdict of Not Guilty.

It is important in cases such as these that the medical man should examine the thighs and vulva of the mother for evidence of wounds, scratches, etc. It is inconceivable that a woman should in efforts at self-delivery inflict *severe* wounds on the child, especially with an instrument of any kind, scissors, knives, etc., without at the same time doing some damage to herself. To excuse severe injuries on the child necessitates the assumption of a condition of frenzy on the part of the mother, and in a frenzy she would probably damage herself. The finding of such lesions on her would strengthen the defence materially.

The following form of violence is very rare. An inquest was held on the newly born child, the daughter of an unmarried woman, who admitted to the police, "Yes, **I threw the baby out of the window.**" That the child was thrown out of an upstairs window there was abundant evidence to show and also that the mother was alone in her confinement.

The medical witness said: On the 23rd January, I was called to the Police Station. I was shown the body of a newly born female child. It was alive. It was well developed but expired shortly after my arrival. It had received no skilled attention at birth, being still covered with sebaceous material. The cord had been torn four and a half inches from its insertion into the child. It had not been tied. The blood was still moist at the severed end. Frothy blood issued from the mouth and nose. Examination showed left thigh was broken at its middle. There was a protrusion of the brain substance and a large quantity of blood from the skull. This had escaped through a rent made in the scalp. *Lungs* were sufficiently inflated nearly to fill the chest cavity. They floated in water with and without the heart. *Heart* and other organs were normal, and had escaped damage. The cause of death was shock, following fracture of the skull and thigh. Verdict: Guilty of the act, but not responsible on account of her mental condition.

Poisoning

This is placed among the possible means of taking a child's life, but we rarely hear of *newly born* children being killed in this way. The earliest age on record where a trial took place in respect of the murder of a child, whose death was caused by poisoning, is two months. Arsenic was given to an infant, and it died in three hours and a quarter after the administration of the poison. If in a case of child murder or of infanticide death from poison is suspected, search must be made for the poison. Some cases have occurred in which children have been wilfully destroyed a week or two after birth by the administration of opium, or excessive doses of purgative medicine. A woman was sentenced to eight years' imprisonment for poisoning her newly born child with concentrated

sulphuric acid. In another case a woman was convicted of poisoning her infant with phosphorus scraped from lucifer matches. In some cases, the poison was found on the napkins used for the child. Stevenson met with an instance of the death of an infant by the administration of chloride of antimony.

In cases in which infants are poisoned there is usually great difficulty in tracing the act of administration to the guilty person. The fluid food given to them renders the admixture of poison easy, and as many persons may have had access to this food, it is often impossible to detect the criminal.

An illegitimate child had been placed out to nurse by its mother. After each visit paid by the mother the child was sick, and after repeated attacks of illness the child died. On inspection, arsenic was found in the body, and this was the cause of death. There was no suspicion against the nurse ; but a strong suspicion fell on the mother. The child was not at any time fed by the mother when she visited it, and the mother had no access to the child's food. No poison could be traced to her possession, and she was never seen giving anything to the infant ; but at each visit she took it in her arms, and was observed to rub its gums with her finger, and soon after each visit sickness followed. There was reason to believe that she had concealed small quantities of arsenic under her finger-nails, and that she had thus administered the poison while rubbing the gums of the child.

If a case arose in which a newly born child had been killed by poison, it would be out of the question to assume that poison given to the child could help the mother's labour ; it would be equally out of the question to imagine that that poison would be given to a child before it was completely born ; and lastly, it could hardly arise accidentally (unless the child were born on to a bag of vermin-killer, etc., of which there would be evidence). The only real difficulty would be that of establishing the fact that poison was the cause of death.

Drowning and Suffocation

The fact of drowning cannot be verified by any appearances in the body of a child which has not breathed. Thus, if a woman caused herself to be delivered in a bath, and the child was forcibly retained under water (a case which is said to have occurred), it would of course die ; but no evidence of the cause of death would be found in the body, unless attempts to respire resulted in the aspiration of water into the air-tubes. After respiration, the signs of drowning will be the same as those met with in the adult (see Vol. I, pp. 542 *et. seq.*). The main question for a witness to decide will be, whether the child was put into the water, or the vessel containing water, living or dead. Infanticide by drowning is by no means common ; the child is generally suffocated, strangled, or destroyed in other ways, and its body is then thrown into water in order to conceal the real manner of its death. The discovery of the dead body of an infant in water must not allow a witness to be thrown off his guard, although a verdict of "found drowned" is so commonly returned in these cases. The body should be carefully inspected, in order to determine what was really the cause of death. All marks of violence on the bodies of children that have died by drowning should be such as to have resulted from accidental causes. The throat and air-passages should be particularly examined.

In a reported case it was clear, from the state of the lungs, that the child had breathed, but no water was found in the lungs or stomach. There was a furrow or depression around the neck, such as would be produced by the pressure of a ligature. It was contended that the child had been strangled and its body thrown into the water after death.

It is not necessary that the *whole* of the body of a child should be submerged in order that it may be destroyed by drowning: the mere immersion of the head in water, or the covering of the mouth with liquid, will suffice to produce the usual effects of asphyxia. **The air-passages should therefore be examined for foreign substances** which may be deposited in them. A woman attempted to kill her child by immersing its head only in a bucket of water. The child was discovered and resuscitated. This method of killing children by drowning may easily deceive a medical man. He would commonly look for evidence of the submersion of the whole body, and if no pond or well were near, he might, from the absence of well-marked *post-mortem* appearances, assign death to some natural cause.

In the case of a woman who drowned her child, but who was acquitted at her trial on the ground that no water had been seen in the room, there were witnesses in court who had sworn before the coroner's jury that water was in a pail in the room, and that it was taken away after the death of the child.

Newly born children may be drowned or suffocated by being thrown into liquid mud or into the soil of a privy. Sometimes the child is killed by other means, and its dead body is thus disposed of for the purpose of concealment. If the child were thrown in living, the phenomena are those of drowning if there is a large quantity of liquid present. The liquid portion of the soil may be found in the air-passages, gullet or stomach, but the mere discovery of soil in the mouth would not suffice to show that the child was living when immersed. The presence of foreign substances, such as dirt, straw, or ashes, in the air-passages, gullet and stomach, must be taken as a proof that the child had been living when immersed in the dirt, etc., and that the substances have been drawn into the passages by inspiration or the act of swallowing.

In *R.* v. *Allridge*, the dead body of a child was found buried in a garden. On examination there was earth in the mouth and throat, as well as in both nostrils; and particles of earth were found in the windpipe and air-tubes, as well as in the stomach. The medical witness referred the death to suffocation, and considered that the earth must have been inhaled. A medical witness for the defence affirmed that the earth might have been carried into the passages of the body accidentally by the percolation of water (in eight days), and that it had not found its way there by inhalation. The accused was acquitted. Although the mouth and throat may thus accidentally receive foreign matters, it is most improbable that the earth should be carried into the air-tubes or stomach by rain-water. The child was probably thrust into the earth when it retained some power of breathing and swallowing, and the earth found in the mouth and throat might be accounted for by the violence with which it was forced into the soil. The nature of the soil, and the circumstances in which the dead body is found, must materially affect a medical opinion in cases of this nature.

On these occasions, the defence may be: (1) That the child was born dead, and that the body was thrown in for concealment: but the evidence may show that the child had breathed, and had probably been born living. (2) It may be alleged that the child breathed for a few moments after birth, but then died, and that the woman thus attempted

to conceal the body. A witness may here be asked whether a woman could have had power to convey the body to the place—a point which must, as a general rule, be conceded. (3) It is frequently asserted that the woman, being compelled to go to the closet, was there *delivered unconsciously* or unexpectedly; that her waters had broken, and that she had no recollection of anything else; or that the child had dropped from her, and was either suffocated or prevented from breathing. All these circumstances may readily occur, but, on the other hand, the explanation may be inconsistent with some of the facts.

The evidence of drowning is practically the same in a newly born infant as in the case of an adult, but it is infinitely more common in the former case for the body to be thrown into the water after death. The stronger the evidence of actual death from drowning (*vide* Vol. I, pp. 546 *et seq.*), especially if the vessel in which the child is drowned is a little way out of reach, the stronger the presumption in favour of a separate existence, because the child must have been carried to the vessel. But this applies only if the foreign material found in the air-passage be not blood, urine, or liquor amnii. Mummification of the cord in the body of a child found genuinely drowned (*vide* p. 161) is practically conclusive proof of a separate existence.

Reference may be made to Vol. I, where homicidal drowning is considered, especially as regards the particular place where the drowning took place.

Deliberate Neglect to tie the Cord

It is impossible for medical evidence to establish whether the cord has been left untied intentionally, though it may be suspected. It is important, however, after noting the method of severance, to ask *why* it was not tied. Ignorance of the necessity for such a procedure may be pleaded, and will be a defence not easily upset, for it is not to be supposed that an ignorant woman has any intuitive knowledge of such necessity.

Burning

If the body of a child is found with burns upon it, the same question will arise as in the case of adults, especially whether they were inflicted before or after death, and whether they were the cause of death (*vide* Vol. I, pp. 489 *et seq.*). If the evidence clearly establishes that they were inflicted before death, then homicide becomes certain, because a fire can never be so near to a mother's vulva that the child's body could be burned in the act of parturition. Hence, in the case of burns there is not so much difficulty in determining whether a criminal act has been committed as in some other forms of violent death.

If merely calcined remains of bones, etc., are laid before a medical witness, he is not likely to be able to do more than to identify them as human remains (*vide* Vol. I, pp. 165 *et seq.*).

The following is a case in point :—

A charwoman was charged with the murder of her newly born child. The charred bones of the child were found in the grate of the cottage occupied by the accused, and there was evidence that the fire had been started by means of paraffin oil, which a midwife purchased while the child was still alive. The defence was that the child was choked by natural causes, and that the accused disposed of the body by burning it because she had no money to meet the burial expenses. The jury acquitted the accused.

CHAPTER XI

UNNATURAL OFFENCES

This subject is of great importance, and bristles with many difficulties. A full consideration, however, would entail excursions into the psychological and sociological aspects of the question which would be out of place in this volume. A relatively brief reference must therefore suffice. The actual offences themselves which will be noticed are :—

Masturbation or onanism.
Indecent exposure.
Sodomy.
Bestiality.
Tribadism (or " Lesbianism ").

On the first of these the law is silent unless the act is done publicly, and so it is on tribadism, but the others are criminal offences.

The statutes dealing with such offences are the Offences against the Person Act, 1861, ss. 61 and 62 :—

Sec. 61. *Whosoever shall be convicted of the abominable crime of buggery, committed either with mankind or with any animal, shall be liable . . . to be kept in penal servitude for life.*

Sec. 62. *Whosoever shall attempt to commit the said abominable crime, or shall be guilty of any assault with intent to commit the same, or of any indecent assault upon any male person, shall be guilty of a misdemeanour, and being convicted thereof shall be liable . . . to be kept in penal servitude for any term not exceeding ten years.*

The Criminal Law Amendment Act, 1885, s. 11 :—

Any male person who, in public or private, commits, or is a party to the commission of, or procures or attempts to procure the commission by any male person of, any act of gross indecency with another male person, shall be guilty of a misdemeanour, and being convicted thereof shall be liable at the discretion of the court to imprisonment for any term not exceeding two years with or without hard labour.

Masturbation. Readers of Krafft-Ebing's " Psychopathia Sexualis " will not need to be informed how the practice of this " crime against self " may easily lead to crime against others. So long as the act is not done openly the law cannot take notice of it. Presumably gross indecency would include only the act done *coram publico* with probable exposure of the person, though it might be made to include a great deal more. When such cases occur they seldom receive notice in the press. Medical evidence could go so far only as to prove the emission of semen ; but unless caught *flagrante delicto* it is not likely that any person would be brought to trial.

Indecent exposure. The exhibition in public of the naked person, or any other act of open and notorious lewdness, is a misdemeanour at common law. Similar exposure, even though in a place of public resort, is not indictable at common law, if it is visible by one person only: but the exposure is indictable, although the place be not one of public resort, if the place be such that a number of persons can and do see the act; for example, on the roof of a house.[1] Bathing in a state of nudity in a place near to which persons frequently pass is a criminal offence.[2]

It has been held that where the prisoner procured the commission of an act of gross indecency with the prisoner himself, the offence was complete.[3]

Medical evidence is incapable of throwing any light upon the actual perpetration of the offence, but it is almost always required in order to ascertain the mental condition of the person who has perpetrated it, and frequently he or she is found to be mentally affected.

It is a felony by statute to commit the abominable crime of **Buggery** which may be defined as the action of a person of the male sex attempting to obtain sexual gratification by means of the anus of a human being (**Sodomy**),[4] or with an animal (**Bestiality**), whether *per vaginam* or *per anum*. Bestiality can be committed by either a man or a woman and with any animal. In both sodomy and bestiality it is sufficient to prove any degree of penetration, though without proof of emission.

The evidence required to establish it is the same as in rape, with two important exceptions: (i) it is not necessary to prove that the offence was committed against the consent of the person upon whom it has been perpetrated; (ii) both parties (if consenting) are equally guilty, except in the case of a boy under the age of fourteen. The guilty associate is a competent witness. In *R.* v. *Wiseman*,[5] a man was indicted for having committed this offence with a woman; and in *R.* v. *Jellyman*,[6] for having committed the offence on his wife.

Unless the person is in a state of insensibility, it is difficult to understand how this offence could be perpetrated upon an adult of either sex against his or her will, inasmuch as the slightest resistance would suffice to prevent its perpetration. The act must be in the part where it is usually committed in the victim or associate of the crime, and if done elsewhere it is not sodomy.[7] If the crime be committed on a boy under fourteen years it is felony in the agent only.[8] Consent is no defence; and a person who consents to the commission of the offence is guilty as a principal unless he be under the age of fourteen.[9] By the Criminal Law Amendment Act, 1922, s. 1, the consent of a young person under the age of sixteen is no defence to a charge or indictment for indecent assault. As a rule, proof of the offence is given without medical evidence, except in the cases of young persons, when marks of physical violence

[1] *R.* v. *Thallman*, 9 Cox, C. C. 388.
[2] *R.* v. *Reed*, 12 Cox, C. C. 1.
[3] *R.* v. *Jones and Bowerbank*, [1896] 1 Q. B. 4.
[4] See Genesis xix., 5.
[5] (1718), Fortes. Rep. 91.
[6] (1838), 8 C. & P. 604. See also *Statham* v. *Statham* [1929] P. 131.
[7] *R.* v. *Jacobs*, Russ and Ry. 331.
[8] *R.* v. *Allen*, 1 Den. 364; and see *R.* v. *Tatam* (1921), 15 Cr. App. Rep. 132.
[9] Cf. *R.* v. *Graham* (1919), 14 Cr. App. Rep. 7.

will generally be apparent. Any degree of penetration is sufficient without proof of emission.[1] In some instances, proof of the perpetration of the crime may be obtained by resorting to microscopical evidence. Stains upon the linen of young persons may thus furnish evidence that the crime has been attempted, if not actually perpetrated. **Pederastia** is that form of buggery in which the passive *rôle* is played by a boy.[2]

Medical Evidence of the Offence

General. It is asserted that those who are in the habit of practising sodomy exhibit certain general characteristics; but as some do not show such stigmata it is unnecessary to enumerate them.

Local. It is not to be expected that any evidence would be found on the penis of the active agent unless it were examined immediately after the act; the only possible evidence obtainable would be the peculiar smell of the anal glands transferred to the penis, and traces of fæces on the organ. The presence of semen is of little importance, for it may be otherwise accounted for, but in any case it is as well to record the fact if fresh seminal matters are found.

Unless an examination is made soon after the perpetration of the crime, the signs of it in the passive agent will disappear. In the case of one long habituated to these unnatural practices, certain changes have been pointed out as medical proofs, among them a funnel-shaped state of parts between the nates, with the appearance of dilatation, stretching, or even a patulous state of the anus, and a destruction of the folded or puckered state of the skin in this part. There may be also marks of laceration, cicatrices, etc., and sometimes the evidence derivable from the presence of syphilitic disease.

This condition of parts would represent the chronic state induced by these practices in the passive agent. In the recent or acute form, fissure and laceration of the sphincter ani, with bruising and effusion of blood, would be found. The appearances above described as belonging to the chronic stage were met with in the case of "Eliza Edwards."

This person was found after death to be a man, although he had passed himself off in dress and habits during life as a woman. On examination of the body there was strong evidence that he had been for many years addicted to unnatural habits. The aperture of the anus was much wider and larger than natural. There was a slight protrusion and thickening of the mucous membrane at the margin. The rugæ or folds of skin which give the puckered appearance to the anal aperture had quite disappeared, so that this part resembled the labia of the female organs. The lining membrane was thickened at the verge of the anus and was in an ulcerated condition. The male organs had been drawn up and secured by a bandage bound round the lower part of the abdomen. A short account of this remarkable case of concealed sex was published in the *Lond. Med. and Physical Jour.*, February, 1833, p. 168.

Tidy adds the following remarks on evidence: "Marks of violence, other than local injuries, are not common in these cases, because the act is usually committed with consent."

There are a few cases where, in a charge of sodomy, stains of semen can constitute important evidence. For instance, seminal stains on the garments of a child too young to have emissions (more particularly

[1] *R.* v. *Cozins*, 6 C. & P. 351.
[2] Priapeia, Beucheler, 1904, 3, p. 139, p. 54; 150.

if they occur on the *posterior* portion of such garment) constitute material evidence. Or again, if they occur on the posterior portion of the shift of a woman who claims to have been unnaturally violated (although in such case corroborative proof is required to render the fact of much avail as evidence), they may prove important in support of the charge. Manifestly, a seminal stain on the garment of an adult male is of no value whatsoever. Seminal matters may be found in the anus and constitute strong evidence of the perpetration of the crime.

At a *post-mortem* in such cases, it will be advisable to note whether there is evidence of the boy or adult having been *gagged*. A gaping anus with a thickened mucous membrane at its margins, and smoothness of the skin around, are characteristics specially to be looked for, whilst chancres or scars of chancres on the mucous membrane of the rectum would be specially significant. It must not be forgotten that dilatation of the rectum and protrusion of the intestines through the anus are common effects of putrefaction.

The crime is not infrequent among seamen. In a case tried at Liverpool, a sailor was proved to have induced a lad to go to sea in order that he might act as the sailor's "passive agent."

The lad was unaware of this, and when the offence was committed, he denounced the accused to the rest of the crew, who complained to the captain, and the accused was handed over to the authorities at a port in South America. The consular authorities inquired into the case, but sent the accused home for trial in England. The boy was examined by Lowndes, who found him suffering from pain in the anus and rectum, although this was some time after the committal of the offence. The accused was convicted, and sentenced to twenty years' penal servitude. In another case where Lowndes gave evidence a blind man was charged with committing this offence upon his own son, a boy of twelve, who was himself the subject of partial paralysis. There were indications that the crime had been committed. The jury found the accused guilty of the attempt, and he was sentenced to ten years' penal servitude.

In *R.* v. *Woods* a blind man was charged with having committed an unnatural offence on his son aged nine years. The jury found the accused guilty of an attempt to commit the offence, and he was sentenced to the maximum term allowed by law—ten years' penal servitude.

Trials for this crime are not infrequent, but the reports of evidence are not made public. *R.* v. *Oscar Wilde* is one of the most notorious, as the accused was for some time a leader of taste and art in certain circles. False charges of this crime are as common as in cases of rape. They are made for the purpose of extortion; and inasmuch as the publication of such a charge, even when unfounded, is greatly dreaded, and in some cases has led to suicide, it often proves a successful method of "blackmail." It is especially deserving of notice that such accusations are frequently made by soldiers and policemen.

In *R.* v. *Boulton and Park* the accused were charged with conspiracy to commit or to incite to the commission of immorality. The accused were young men who had for some time frequented public places dressed as women, and had been seen to associate with men as if they were women of the town. They were beardless youths, and one of them (Boulton) had a countenance so feminine that when seen by the medical examiners he appeared like a young woman in man's clothing. When dressed as fashionable women, they imposed upon all who saw them. These practices had continued at intervals for one or two years before they were detected and exposed. The defence was that they had dressed themselves as women for the purpose of performing at private theatricals, but this did not account for all

their activities or for their correspondence with many persons who were believed to be accomplices. They also assumed female names, and used them in correspondence with men. "They habitually walked the streets and frequented places of public amusement in women's clothes, practising all the petty arts of prostitutes, submitting to be entertained as such by gentlemen, and then suddenly resuming the privileges of their own sex." After a lengthy trial the jury returned a verdict of not guilty. The medical opinions differed, but there was no definite evidence that any unnatural offence had been committed.

Occasionally schoolmasters and scoutmasters are charged with criminal offences in connexion with boys in their charge. In many such cases mutual or one-sided masturbation is practised.

In *R.* v. *Smith and another*. Smith was sentenced to penal servitude for life, and the other prisoner to ten years' penal servitude. Smith (aged thirty-five) was a thick-set muscular man, with dark bushy beard and whiskers. He was not at all feminine in appearance, but was dandified in dress, and was known as "Captain Smith." He had debauched about fifty Post Office lads. His practice was to enter into conversation with the lads, to give them wine, suppers, spirits, cigars, etc., to show them indecent pictures, and finally to commit unnatural crimes upon them. No medical evidence was given.

The following remarks are by Sutherland :—

"The Signs of Passive Pæderasty. Into the description of these Tardieu and others have gone at considerable length, with the result that to many minds the 'infundibulum' and the 'triangular sodomitic wound' are a *sine qua non* of passive pæderasty. Undoubtedly Tardieu and his school did see what they describe, and where these signs are present the evidence is complete ; but where they are absent the innocence of the accused should not be presumed in all cases. Witness the following case :—A Brahmin, aged about forty, sought treatment for what he said was a boil on the perinæum. On examining the 'boil,' I found it to be a typical Hunterian chancre, situated one inch in front of the anus, and on being questioned the patient admitted that he might have contracted it from one of his friends. He volunteered the statement that he had been a pathic for at least twenty years, so I examined him for the classical signs of his aberration, and found none of them. The genitals were well formed, there was no deformation of the anal region, no infundibulum nor loss of rugæ, and the tone of the sphincter was normal."

Bestiality (see above under "Buggery"). Trials for this crime committed upon animals, such as cows, mares, and she-asses, are not unknown at the assizes. They are not reported ; and therefore do not attract any public notice. The criminals are usually youths or men employed to look after the animals. In most of these cases the criminal has been caught *flagrante delicto*, or in such circumstances as to leave no doubt of the attempt, if not of the completion, of the act of unnatural intercourse.

Medical evidence is seldom required to sustain the prosecution. There may, however, be circumstances which can be properly interpreted only by an expert. The hair of the animal may be found on the perpetrator, or marks of blood or feculent matter upon his clothing ; and in such cases analysis, or the microscope, may enable a medical witness to express an opinion in proof or disproof of the charge. It would obviously be more important to discover animal's hair on the underclothing than on coat, trousers, etc.

In one case tried at the assizes, where a man was charged with having had unnatural intercourse with a cow, the prosecution was able to show that some short coloured hairs found on the prisoner's person resembled those of the animal. In another case Stevenson found the peculiar coloured hairs of a mare upon the prisoner's clothes, and spermatozoa on his trouser-flap.

The medical jurists of Germany have taken a great interest in cases of sodomy and bestiality ; and in some of their reports they have contrived to throw an air of science over the details of this detestable crime. In the following German case a sub-officer was charged by his captain with unnatural intercourse with a mare, and in support of the charge microscopical evidence was given by a medical witness.

The captain, on entering the stable suddenly, found the accused in the act of moving away from the stall of the animal. Upon examining the mare some small abrasions were found about the genitals, and a slight escape of bloody mucus from these parts. The accused willingly submitted himself to examination, and some stains of blood were found on his shirt ; and on the penis between the prepuce and the glans there were a number of short, dark, pointed hairs. The accused accounted for them by saying that the night before he had had connection with a woman. The hairs were carefully examined by microscope, and they were found to be shorter, thicker, and more pointed than those of a human being. They were also coarse, and less transparent. Comparing them with hairs gently rubbed off the back part of the mare, they exactly corresponded in colour, form, and length, so as to leave no doubt in his mind that there had been unnatural intercourse. The species from which the blood on the shirt was derived was not established. That, however, was not a necessary part of the evidence.

Tribadism or "Lesbianism" means the gratification of the sexual desire of a woman by another woman. It is not an offence known to the criminal law of England ; but see the *dictum* of Avory, J., as to the capacity of one woman to commit an indecent assault on another woman.[1]

The vice is usually practised by women who are suffering from nymphomania, and an allegation in regard thereto should suggest the desirability of holding an inquiry into the woman's mental state.[2]

Sexual passion or desire is stronger in some people (of both sexes) than in others ; and wherever a charge of unnatural sexual gratification is brought against a person, a careful inquiry should be made into the mental, moral and social environment.

Further, abnormal sexual passion is frequently evidence of insanity, and the victim of it may be treated more properly in a mental hospital than by imprisonment amongst ordinary criminals.

Medical experience shows that bodily conditions may account for pathological increase in sexual desire and passion, which may lead to crime. Such a condition may take the form either of lack of balance in the secretion of the sex glands and other ductless glands, or of actual disease (*Herpes preputialis*, for example) capable or not of cure. In older men the condition known as "enlarged prostate" is certainly a direct provocative, in many cases, to the commission of sexual crime. The latter condition can hardly be urged as an excuse, but it may be offered as an explanation, and inasmuch as it is a condition which is capable of cure by operation, it may be considered right to offer to an accused man the alternative of submitting to cure by operation before sentencing him, the results of operation to be awaited before sentence is passed.

[1] *R.* v. *Hare*, [1934] 1 K. B., 354.
[2] Th. Gautier, "Mad. de Maupin," end.

PART II

POISONING AND TOXICOLOGY

This large and very important section of forensic medicine may be conveniently divided into the following subsections, *viz.* :—

The Law on Poisons, including the definition of a Poison or Noxious Thing.

Dangerous Drugs.

How Poisons Act, and the Circumstances which influence their Action.

The Diagnosis of Poisoning.

What to do in Cases of (suspected or proved) Poisoning.

Poisoning by the various (Groups of) Individual Poisons.

CHAPTER XII

The Law of Poisons, including the Definition of a Poison or Noxious Thing

Offences against the Person Act, 1861, ss. 11 to 25.

Whosoever shall administer to, or cause to be administered to or taken by any person any poison or other destructive thing, with intent to commit murder, shall be guilty of felony (sect. 11).

Whosoever shall attempt to administer to or shall attempt to cause to be administered to, or to be taken by any person, any poison or other destructive thing, with intent to commit murder shall be guilty of felony (sect. 14).

Whosoever shall, by any means other than those specified in any of the preceding sections of this Act, attempt to commit murder, shall be guilty of felony (sect. 15).

Whosoever shall unlawfully apply or administer to or cause to be taken by, or attempt to apply or administer to or attempt to cause to be administered to or taken by, any person, any chloroform, laudanum, or other stupefying or overpowering drug, matter, or thing, with intent in any of such cases, thereby to enable himself or any other person to commit, or with intent thereby to assist any other person in committing, any indictable offence, shall be guilty of felony (sect. 22).

Whosoever shall unlawfully and maliciously administer to, or cause to be administered to or taken by, any other person any poison or other destructive or noxious thing, with intent to injure, aggrieve, or annoy such person, shall be guilty of a misdemeanour (sect. 24).

If, upon the trial of any person charged with the felony above mentioned, the jury shall not be satisfied that such a person is guilty thereof, but shall be satisfied that he is guilty of the misdemeanour above mentioned, then, and in every case, the jury may acquit the accused of such felony and find him guilty of such misdemeanour (sect. 25).

Definition of a Poison. Before the words "destructive" and "noxious thing" were introduced into the law, the definition of what was and what was not a poison was a matter of the very highest importance. Now the definition is chiefly of historical interest, except in so far that a cross-examining counsel may legitimately ask a medical witness for a definition with a view to showing either bias or ignorance in the witness's mind. An attempt must therefore be made to suggest a sufficient definition.

A poison is commonly defined to be a substance which, when administered or taken *in small quantity*, is capable of acting deleteriously on the body. In popular language, this term is applied only to those substances which in small doses cause death. This popular view of the nature of a poison is too restricted for the purposes of medical jurisprudence. It would obviously exclude numerous compounds the poisonous properties of which cannot be disputed, as, for example, the salts of copper, tin, zinc, lead, and antimony. These, generally speaking, act as poisons only when administered in quantities which in a *medicinal* sense would be large doses. Some substances, such as nitre or chloride of sodium (common salt), have not been observed to have a noxious action except when taken in quantities which in *popular* language would be called large, while arsenic, on the contrary, acts as a poison in small doses; but in the medico-legal view, whether a man dies from the effects of an ounce of nitre or two grains of arsenic, the responsibility of the person who criminally administers the substance is the same. Each may be regarded as a poison, differing from the other only in its degree of activity and in its mode of operation. The result is the same: death is caused by the substance taken; and the *quantity* required to cause death, even if it could be always accurately determined, cannot enable us to distinguish a poisonous from a non-poisonous substance. If, then, a medical witness be asked, "What is a poison?" he must beware of adopting this popular definition, or of confining the term poison to a substance which is capable of operating as such in a small dose taken once.

In legal medicine, it is difficult to give such a definition of a poison as shall be entirely free from objection. Perhaps the most comprehensive which can be suggested is this:—"A poison is a substance which, when taken into the mouth or stomach or when absorbed into the blood, is capable of seriously affecting health or of causing death by its action on the tissues with which it immediately, or after absorption, comes in contact." Poisons enter the blood by various channels: some take the form of gases or vapours; these operate rapidly through the lungs. Others are liquid or solid, and these may reach the blood either through

the skin or through a wound, but usually through the lining-membrane of the stomach or bowels, as when they are taken or administered in the ordinary manner. The latter chiefly give rise to medico-legal investigations. Some substances act as poisons by any one of these channels; thus arsenic is a poison whether it enters the blood through the lungs, the skin, or the stomach and bowels: but such poisons as those of snakes, or rabies, and of glanders, appear greatly to affect the body only through a wound in the skin. When introduced into the stomach, these animal poisons have been found to be almost inert. This subject will be further considered (*vide* "Animal Poisons").

It will be noticed that this definition certainly includes the great class of corrosives and also most of the mechanical bodies, such as powdered glass.

It is not possible in the abstract to define the boundary between a medicine and a poison. It is often stated that a medicine in a large dose is a poison, and that a poison in a small dose is a medicine; but a medicine such as tartrate of antimony may be easily converted into a poison by giving it in small (medicinal) doses at short intervals, either in states of the body not adapted to receive it or in cases in which it exerts an injuriously depressing effect. Some deaths have been occasioned by this wilful misuse of antimony in doses which might be described as *medicinal*, although in the cases referred to no other intention could have existed, in the secret administration of this substance, than that of destroying life. A person may die either from a large dose of a substance given at once or from a number of small doses given at such intervals that the system cannot recover from the effects of one before another is administered. This remark applies to a great number of medicines which are not commonly included in a list of poisons. In fact, the only real difference between a medicine and a poison is the *intent* with which they are purposely, not accidentally, given, the one to save life or cure disease, the other to destroy life or simulate disease.

Definition of Noxious Thing. It is impossible to lay down any abstract rules as to what is a noxious thing within the meaning of the Offences against the Person Act, 1861. The medical witness must be prepared to show that *in the circumstances* the "thing" was "noxious." In *R.* v. *Hennah*,[1] it was decided that, in order to constitute the offence of administering "a certain destructive and noxious thing," the thing administered must be noxious *in itself*, and not merely when taken in excess; although it may have been administered with intent to injure or to annoy. Moreover, judges exercise a certain discretion in deciding between a substance which is essentially noxious and one which is noxious only by reason either of its excess or from the circumstances attending its exhibition.

Further, the substance administered may not be a poison in the medical signification of the term, and may not be popularly considered as such; yet, when taken, it may be noxious to health or destructive to life. Examples of substances of this description are iron filings, powdered glass, diamond dust, sponge, pins and needles, and similar bodies, which have been administered with the wilful intent of injuring,

[1] 13 Cox, C. C. 547.

and have, on various occasions, given rise to criminal proceedings. In cases of this kind, the guilt of a prisoner may often depend on the meaning assigned by a medical witness to the words *destructive thing*. For example, liquid mercury may be poured down the throat of an infant with the deliberate intention of causing death. In such circumstances a question of a purely medical nature would arise whether mercury be a "destructive thing" or not; and the conviction of a prisoner would probably depend on the answer.

Intent. The intent with which a thing has been applied or administered is an essential element in law. Whether the administration be followed by any bodily injury or not, the act is a felony or misdemeanour, provided that intent to commit murder or to injure or to annoy can be proved.

If a noxious thing is administered which is likely to occasion death, and the person who administers it is indifferent whether death results or not, such person is to be regarded as having contemplated the probable results of his own action. In *R.* v. *Wilkins* a man administered cantharides to a woman with intent to excite her sexual passions in order that he might have connection with her.[1]

In a case where a medical man was charged with "attempting to cause to be administered" a poisonous dose of laudanum to an infant, a woman who nursed the child stated that the accused delivered to her two bottles containing a brown liquid, labelled "One teaspoonful every three hours," and directed her to give it to the child. None was given. Some months after the death of the child from natural causes, the two bottles still full of liquid were produced as evidence against the accused. The prescribed dose contained about five minims of laudanum, or nearly one half-grain of opium—a dose likely to prove fatal to an infant only a month old. Assuming the truth of the statement of the woman who brought the charge, the only inference to be drawn from the prescription of such a dose for an infant by a medical man would be that he intended to kill the child. The charge was dismissed, as the woman who brought it was not to be believed on her oath, and had brought it with a "blackmailing" object.

Poisons are sometimes mixed with food, and thus administered with a view to injuring or annoying a person.

(*a*) In one case eight members of a family suffered from severe symptoms of poisoning by reason of the wanton administration of cantharides.

(*b*) In another case several members of a family suffered from severe sickness as a result of tobacco having been put into water contained in a tea-kettle.

(*c*) Cases are known where tartar emetic has been dissolved in beer or liquids as a mere frolic, without any proved or probable intention on the part of the offender to destroy life. *M'Mullen's* case revealed the existence in the north of England of an extensive system of poisoning in which tartar emetic was the substance employed. This drug, mixed with cream of tartar, was openly sold by druggists under the name of "quietness powders," to women who administered the powders to their husbands with a view to curing them of habitual drunkenness.

The **quantity** of a poisonous substance found in an article of food, or in a dead body, **does not affect the culpability** of a person indicted for administering it. In *R.* v. *Hartley*, where an attempt was made to administer sulphuric acid mixed with coffee, the judge said: "If poison be administered with intent to murder, it is not necessary that there should be enough in the article administered to cause death. If

[1] 9 Cox, C. C. 20.

any poison be there, and the intent be proved, the crime of attempting to administer poison is complete." A similar decision was given in *R.* v. *Bacon*. In *R.* v. *Southgate*, the judge said, in reply to an objection taken, that it was quite immaterial to define or to prove in what vehicle a poison was given, or whether it was administered in a solid or liquid state.

In *R.* v. *Armstrong*,[1] the Court of Criminal Appeal held that evidence that the prisoner had, subsequently to the death of the victim, attempted to poison another person by administering arsenic in a buttered scone at tea, was admissible to rebut a defence that the deceased had committed suicide, and (*semble*) thus to show felonious intent.

It is clear that medical evidence cannot have any direct bearing on intent ; but, indirectly, it may have a bearing which is conclusive, for example, where a dose of five minims of laudanum was administered to an infant. In this way, drugs perfectly innocuous in themselves, doses well within medicinal limits, methods quite appropriate in other circumstances, may all be used with criminal intent ; and medical evidence may be the only means of proving intent by showing that the factor employed was inappropriate for the particular case in which it was utilised.

As to intent, the law does not regard **the manner** in which the substance administered acts. If it be capable of causing death or of injuring health, it is of little importance, so far as the responsibility of a prisoner is concerned, whether its action on the body is of a mechanical or of a chemical nature, or whether it operates fatally by absorption into the blood or not. Thus, a substance which acts mechanically on the stomach or bowels, may, if wilfully administered with intent to injure, involve a person in a criminal charge as much as if arsenic or one of the ordinary poisons had been administered. If the substance criminally administered destroys life, whatever may be its nature or mode of operation, the accused is charged with murder or manslaughter ; and the duty of a medical witness consists in showing that the substance taken was a certain cause of death. If, however, death be not the consequence, then the accused may be tried for the attempt to murder or to annoy by poison. The words of the statutes are general, and include all kinds of substances, whether they are popularly or professionally regarded as poisons or not.

This question of " intent " may present itself under another aspect. In *R.* v. *Cluderay*, the accused was found guilty of having administered to a child nine weeks old two *cocculus indicus* berries. The child vomited one of them, and the other passed through her body in the course of nature. The *cocculus indicus* was rightly regarded as a poison. The poison consists in the presence of a glucoside, which is extracted from the kernel ; all the noxious properties are in the kernel ; it has a very hard exterior or pod, which requires much force to break.

The Pharmacy and Poisons Act, 1933

This Act (which replaced earlier enactments having a similar purpose), and the Statutory Rules made thereunder, control the supply and purchase of a large number of substances which are regarded and defined as poisons

[1] [1922] 2 K. B. 555.

within the meaning of the Act. The Home Secretary, advised by a body known as the Poisons Board, maintains a list of the substances which are to be so regarded, and makes rules defining the extent of the restrictions which shall apply to each. This Poisons List is divided into two parts, namely,

PART I, consisting of those poisons which may be sold only by authorised sellers of poisons (that is to say by registered pharmacists) ;

PART II, consisting of those poisons which may be sold, not only by registered pharmacists, but by listed sellers of Part II poisons, meaning traders who have been authorised to sell such poisons by virtue of their names having been entered on a list maintained for this purpose by a Local Authority.

In general, it may be said that those poisons required in the treatment of human ailments appear in Part I of the list, while those in common use for domestic or commercial purposes appear in Part II. The list has been revised from time to time, its present composition being as follows :—[1]

PART I

Acetanilide ; alkyl acetanilides

Alkali fluorides other than those specified in Part II of this List

Alkaloids, the following ; their salts, simple or complex :—

- Acetyldihydrocodeinone ; its esters
- Aconite, alkaloids of
- Apomorphine
- Atropine
- Belladonna, alkaloids of
- Benzoylmorphine
- Benzylmorphine
- Brucine
- Calabar bean, alkaloids of
- Coca, alkaloids of
- Cocaine
- Codeine
- Colchicine
- Coniine
- Cotarnine
- Curarine
- Diacetylmorphine
- Dihydrocodeinone ; its esters
- Dihydrohydroxycodeinone ; its esters
- Dihydromorphine ; its esters
- Dihydromorphinone ; its esters
- Ecgonine ; its esters
- Emetine
- Ephedra, alkaloids of
- Ergot, alkaloids of
- Ethylmorphine
- Gelsemium, alkaloids of
- Homatropine
- Hyoscine
- Hyoscyamine
- Jaborandi, alkaloids of
- Lobelia, alkaloids of

Alkaloids—*continued*

- Morphine
- Papaverine
- Pomegranate, alkaloids of
- Quebracho, alkaloids of, other than the alkaloids of red quebracho
- Sabadilla, alkaloids of
- Solanaceous alkaloids not otherwise included in this List
- Stavesacre, alkaloids of
- Strychnine
- Thebaine
- Veratrum, alkaloids of
- Yohimba, alkaloids of

Allylisopropylacetylurea

Amidopyrine ; its salts

Amino-alcohols, esterified with benzoic acid, phenylacetic acid, phenylpropionic acid, cinnamic acid or the derivatives of these acids

Amyl nitrite

Antimony, chlorides of ; oxides of antimony ; sulphides of antimony ; antimonates ; antimonites ; organic compounds of antimony

Arsenical substances, the following, except those specified in Part II of this List : arsenic, halides of ; oxides of arsenic ; arsenates ; arsenites ; organic compounds of arsenic

Barbituric acid ; its salts ; derivatives of barbituric acid ; their salts ; compounds of barbituric acid, its salts, its derivatives, their salts, with any other substance

[1] Poisons List Confirmation Order, 1935, as amended by Poisons List (Amendment) Orders), 1937, 1938 and 1940.

Barium, salts of, other than barium sulphate and the salts of barium specified in Part II of this List

Beta-aminopropylbenzene; its salts; its N-alkyl derivatives; their salts; beta-amino-iso-propylbenzene; its salts; its N-alkyl derivatives; their salts

Butyl chloral hydrate

Cannabis (the dried flowering or fruiting tops of *Cannabis sativa* Linn.); the resin of cannabis; extracts of cannabis; tinctures of cannabis; cannabin tannate

Cantharidin; cantharidates

Chloral formamide

Chloral hydrate

Chloroform

Creosote obtained from wood

Croton, oil of

Digitalis, glycosides of; other active principles of digitalis

Dinitrocresols; dinitronaphthols; dinitrophenols; dinitrothymols

Elaterin

Ergot (the sclerotia of any species of *Claviceps*); extracts of ergot; tinctures of ergot

Erythrityl tetranitrate

Glyceryl trinitrate

Guanidines, the following: polymethylene diguanidines, dipara-anisylphenetyl guanidine

Hydrocyanic acid; cyanides; double cyanides of mercury and zinc

Insulin

Lead acetates; compounds of lead with acids from fixed oils

Mannityl hexanitrate

Mercury, oxides of; nitrates of mercury; mercuric ammonium chlorides; potassio-mercuric iodides; mercuric oxycyanides; mercuric thiocyanate

Metanitrophenol; orthonitrophenol; paranitrophenol

Nux Vomica

Opium

Orthocaine; its salts

Ouabain

Oxalic acid

Oxycinchoninic acid, derivatives of; their salts; their esters

Para-aminobenzenesulphonamide; its salts; derivatives of para-aminobenzenesulphonamide having any of the hydrogen atoms of the para-amino group or of the sulphonamide group substituted by another radical; their salts

Para-amino-benzoic acid; esters of; their salts

Phenetidylphenacetin

Phenols (any member of the series of phenols of which the first member is phenol and of which the molecular composition varies from member to member by one atom of carbon and two atoms of hydrogen) except in substances containing less than sixty per cent, weight in weight, of phenols; compounds of phenol with a metal, except in substances containing less than the equivalent of sixty per cent, weight in weight, of phenols

Phenylcinchoninic acid; salicyl-cinchoninic acid; their salts; their esters

Phenylethylhydantoin; its salts; its acyl derivatives; their salts

Phosphorus, yellow

Picric acid

Picrotoxin

Pituitary gland, the active principles of

Savin, oil of

Strophanthus; glycosides of strophanthus

Sulphonal; alkyl sulphonals

Suprarenal gland, the active principles of; their salts

Thallium, salts of

Thyroid gland, the active principles of; their salts

Tribromethyl alcohol

Part II

Ammonia

Arsenical substances, the following:—

- Arsenic sulphides
- Arsenious oxide
- Calcium arsenates
- Calcium arsenites
- Copper acetoarsenites
- Copper arsenates
- Copper arsenites

Arsenical Substances *continued*

- Lead arsenates
- Potassium arsenites
- Sodium arsenates
- Sodium arsenites
- Sodium thioarsenates

Barium, salts of, the following:—

- Barium carbonate
- Barium silicofluoride

Formaldehyde

Hydrochloric acid

Hydrofluoric acid ; potassium fluoride ; sodium fluoride ; sodium silicofluoride

Mercuric chloride ; mercuric iodide ; organic compounds of mercury

Metallic oxalates

Nicotine ; its salts

Nitric acid

Nitrobenzene

Phenols as defined in Part I of this List in substances containing less than sixty per cent, weight in weight, of phenols ; compounds of phenol with a metal in substances containing less than the equivalent of sixty per cent, weight in weight, of phenols

Phenylene diamines ; toluene diamines ; their salts

Potassium hydroxide

Sodium hydroxide

Sulphuric acid

(*Note.* Several poisons in this list are exempted by the Poisons Rules (Rule 11 and Third Schedule) made by the Secretary of State under the Pharmacy and Poisons Act, 1933, from the application of the Act when present in certain specified substances or articles.)

All poisons included in the list are not subject to the same restrictions. The detailed measures of control for individual poisons or groups of poisons are defined by the Home Secretary in the Poisons Rules which are made by him under the Pharmacy and Poisons Act, 1933.[1] In this way, the various poisons are allocated to one or other of a series of Schedules—eight in number—according to the nature and extent of control which is considered advisable, or, it may be, the extent to which they may be exempted from control.

Most of the well-recognised poisons are included in the *First Schedule*, the poisons in which are subjected to the greatest restrictions. A First Schedule poison, whether it be from Part I or Part II of the Poisons List, may not be sold unless the purchaser is known, or adequately introduced to the seller ; unless the purchaser is regarded by the seller as a person to whom the poison may properly be sold ; unless the purchaser states a purpose for the poison which the seller believes to be genuine and legitimate ; unless the purchaser affixes his signature to a record of the transaction in a book kept for the purpose. Furthermore, the seller must observe certain statutory regulations regarding labelling and transmission by post.

It is to be noted that these restrictions do not mean that access to First Schedule poisons is confined to any particular section of the community, e.g., doctors, dentists, etc., and a medical prescription is not necessary. If First Schedule poisons are prescribed, no special regulations apply to such prescriptions.

In the *Fourth Schedule*, however, there are listed a number of drugs which may be supplied only on a prescription from a medical practitioner, a dentist, or a veterinary surgeon. The prescription must conform with the requirements of the Act in certain particulars, e.g., it must be dated and signed (not initialed) by the prescriber, and it must include his own address as well as the name and address of the person for whom the medicine is prescribed. The prescription may not be dispensed more than once unless there are instructions to the contrary. The drugs in the Fourth Schedule are not of the type popularly regarded as poisonous.

[1] For a proper understanding of the scope and intention of the Act, it is essential that the Act itself and the rules made thereunder should be studied. Readers will also find it helpful to refer to a text-book dealing more exclusively and extensively with the subject of Forensic Pharmacy.

but they are potentially dangerous if taken without adequate medical supervision. It should be noted, however, that they are listed as Part I poisons and that they are also included in the First Schedule. The Fourth Schedule poisons are :—

(1) Amidopyrine ; its salts ;

(2) Barbituric acid ; its salts ; derivatives of barbituric acid ; their salts ; compounds of barbituric acid, its salts ; its derivatives, their salts, with any other substance ;

(3) Dinitrocresols ; dinitronaphthols, dinitrophenols ; dinitrothymols;

(4) Para-aminobenzenesulphonamide ; its salts ; derivatives of para-aminobenzenesulphonamide having any of the hydrogen atoms of the para-amino or of the sulphonamide group substituted by another radical ; their salts ;

(5) Phenylcinchoninic acid ; salicyl-cinchoninic acid ; their salts ; their esters ;

(6) Sulphonal ; alkyl sulphonals.

The remaining schedules define a variety of other restrictions or special provisions which are of interest to all sellers of poison, authorised or listed, but which have little importance from a wider medico-legal viewpoint.

Under a special rule, the retail sale of *strychnine* is prohibited, except as a medicinal ingredient, or for purposes of research, chemical analysis or scientific education, or, under special authority, for the killing of moles and seals.

CHAPTER XIII

DANGEROUS DRUGS

The Dangerous Drugs Acts, 1920—32, regulate the importation, exportation, manufacture, sale, and use of opium and certain other drugs which are regarded as "dangerous" because they tend to cause drug addiction. Almost without exception, the drugs and their preparations which are covered by the Dangerous Drugs Acts are also Part I and First Schedule poisons. The object of these Acts and of the statutory regulations made thereunder is to prevent traffic in dangerous drugs, and to endeavour to keep the use of such drugs entirely under the control of the medical profession. The substances to which the Acts apply are set out in sect. 1 of the Dangerous Drugs Act, 1932, sub sect. (1) of which is as follows :—

"(1) *The drugs to which this Part of this Act applies are—*

(*a*) *Medicinal opium ;*

(*b*) *Any extract or tincture of Indian hemp ;*

(*c*) *Morphine and its salts, and diacetylmorphine (commonly known as diamorphine or heroin) and the other esters of morphine and their respective salts ;*

(*d*) *Cocaine (including synthetic cocaine) and ecgonine and their respective salts, and the esters of ecgonine and their respective salts ;*

(*e*) *Any solution or dilution of morphine or cocaine or their salts in an inert substance whether liquid or solid, containing any proportion of morphine or cocaine, and any preparation, admixture, extract or other substance (not being such a solution or dilution as aforesaid) containing not less than one-fifth per cent. of morphine or one-tenth per cent. of cocaine or of ecgonine ;*

(*f*) *Any preparation, admixture, extract or other substance containing any proportion of diacetylmorphine ;*

(*g*) *Dihydrohydroxycodeinone, dihydrocodeinone, dihydromorphinone, acetyldihydrocodeinone, dihydromorphine, their esters and the salts of any of these substances and of their esters, morphine-N-oxide (commonly known as genomorphine), the morphine-N-oxide derivatives, and any other pentavalent nitrogen morphine derivatives ;*

(*h*) *Thebaine and its salts, and (with the exception of methyl-morphine, commonly known as codeine, and ethylmorphine, commonly known as dionin, and their respective salts) benzylmorphine and the other ethers of morphine and their respective salts ;*

(*i*) *Any preparation, admixture, extract or other substance containing any proportion of any of the substances mentioned in paragraph (g) or in paragraph (h) of this sub section.*"

For the purpose of the foregoing provision the expression "ecgonine" means lævo-ecgonine and includes any derivatives of ecgonine from which it may be recovered industrially, and the percentage in the case of morphine shall be calculated as in respect of anhydrous morphine.

Persons authorised to possess these drugs include duly qualified medical practitioners, registered dentists, registered veterinary surgeons, and pharmacists, who are employed or engaged in dispensing medicines at a public hospital or other public institution.

The medical practitioner is required to keep and to preserve for two years prescriptions, records and registers, etc., of all such drugs purchased and used in his practice.

The most important of the regulations are as follows :—

1. The prescription may be an ordinary prescription or a National Health Insurance form.

2. It must be in writing and signed by the person giving it with his usual signature, and dated by him.

3. Except in the case of a Health Insurance prescription it must specify the address of the person giving it.

4. It must specify the name and address of the patient in full.

5. If it prescribes a preparation contained, or compounded of preparations all of which are contained, in the British Pharmacopœia, the British Pharmaceutical Codex, or the Drug Tariff issued by the Minister of Health for National Health Insurance purposes, it must specify the total amount of the preparation, or of each preparation, as the case may be, and in any other case, it must specify the total amount of the drug to be supplied.

The chief duties of the pharmacist or dispenser who supplies a drug or preparation on a prescription are that he must not supply a drug or preparation on such prescription unless the prescription comply with the foregoing requirements ; and unless (i) in the case of a health insurance prescription he has no reason to suppose that the prescription is not genuine ; or (ii) in the case of any other prescription he *either* (a) is acquainted with the signature of the person by whom it purports to have been given, and has no reason to suppose that it is not genuine ; *or* (b) has taken reasonably sufficient steps to satisfy himself that it is genuine.

The prescription must *not* be dispensed *more than once*, unless the prescription authorises repetition at repeated intervals, but in all cases the *total* number of dispensings must *not exceed three*.

The pharmacist or dispenser must retain possession of the prescription, and keep it on the premises where it was dispensed, unless it is a Health Insurance prescription. In no circumstances must the patient be allowed to have possession of it.

A medical practitioner need not make any record of any of the above-mentioned drugs, or of their salts, provided the drugs are administered to the patient in his presence. Medical practitioners may write prescriptions for themselves.

Where morphine or a similar drug is prescribed for a patient regularly, the medical practitioner should be careful to protect himself from a

charge of improperly administering dangerous drugs by consulting with another medical practitioner.

It has been ruled that—

(i) A medical practitioner (but *not* a dentist) may *dispense* dangerous drugs for the use of his patients ;

(ii) Typewritten prescriptions may be accepted as " written " ;

(iii) Telephoned orders sent by a medical practitioner to a pharmacist in respect of drugs to be supplied to himself may be executed, but a written order must follow within twenty-four hours ;

(iv) A foreign medical practitioner, unless registered in England, is not a medical practitioner within the meaning of the Dangerous Drugs Acts.

Where the Home Secretary has reason to suspect that a medical practitioner or dentist is supplying or prescribing drugs or preparations to or for himself or any other person otherwise than is properly required for the medical or dental treatment of himself or that other person, the Home Secretary may refer the matter to a special " Reference Tribunal " referred to in the Dangerous Drugs (Consolidation) Regulations, 1928.

The following preparations which contain opium or morphine are excluded from the regulations, so that prescriptions for these are not subject to any restrictions :—

Cereoli iodoformi et morphinæ, B.P.C.
Emp. opii, B.P. 1898.
Lin. opii, B.P. 1914.
Lin. opii ammon., B.P.C.
Pasta arsenicalis, B.P.C.
Pil. hydrarg. c. opio, B.P.C.
Pil. ipecac. c. scilla, B.P. 1914.
Pil. plumbi c. opio, B.P. 1914.
Pil. digitalis et opii co., B.P.C.
Pil. hydrarg. c. cret. et opii, B.P.C.
Pulv. cretæ aromat. c. opio, B.P. 1932.
Pulv. ipecac. co., B.P. 1914.
Pulv. ipecac. et opii, B.P. 1932.
Pulv. kino co., B.P. 1914.
Suppos. plumbi co., B.P. 1914.
Suppos. plumbi c. opio, B.P. 1932.
Tablettæ plumbi c. opio, B.P.C.
Ung. gallæ c. opio, B.P. 1914.
Ung. gallæ co., B.P.C.
Elixir diamorphinæ et terpini c. apomorphina, B.P.C.
Linctus diamorphinæ Camphoratus, B.P.C.
Linctus diamorphinæ c. ipecacuanha, B.P.C.
Linctus diamorphinæ et scillæ, B.P.C.
Linctus diamorphinæ et thymi, B.P.C.

Mixtures of emp. opii, B.P. 1898 with other plasters of the British Pharmacopœia 1914 and 1932, and of the British Pharmaceutical Codex.

Mixtures of lin. opii, B.P. 1914 with other liniments of the British Pharmacopœia 1914 and 1932, and of the British Pharmaceutical Codex.

Mixtures of lin. opii ammon., B.P.C. with other liniments of the British Pharmacopœia 1914 and 1932, and of the British Pharmaceutical Codex.

Mixtures of pulv. ipecac. co., B.P. 1914 and of pulv. ipecac. et opii, B.P. 1932, with any of the following :—

Hydrarg. c. cret. B.P. 1914 and 1932.
Acetylsalicylic acid.
Phenacetin.
Quinine and its salts.
Sodium bi-carbonate.

Mixtures of ung. gallæ c. opii, B.P. 1914 and of ung. gallæ co., B.P.C. with other ointments and plasters of the British Pharmacopœia 1914 and 1932, and of the British Pharmaceutical Codex.

It should specially be noted that if any other medicament is mixed with any of the above-mentioned preparations, and the percentage of "dangerous" drug exceeds the legal limit, the Home Office authorities regard the resulting product as coming within the regulations.

The following preparations are outside the regulations by reason of their being below the legal limit :—

Tr. camph. co.
Tr. chlorof. et morph. (chlorodyne, B.P. 85).
Tr. opii ammon.
Troch. morph. et ipecac.

The Therapeutic Substances Act, 1925

This Act regulates the manufacture, sale, and importation of therapeutic substances. No person (other than a medical practitioner preparing for his own patient or for another practitioner for an individual patient) may manufacture these substances (which include vaccines, sera, toxins, salvarsan, insulin) except under licence from the Ministry of Health. The detailed standards are contained in the Therapeutic Substances Regulations, 1931. The Therapeutic Substances Act also applies to penicillin.

The Penicillin Act, 1947

Under this Act, Penicillin and its preparations may be supplied only on a signed and dated prescription by a registered medical practitioner. Unless it is otherwise specified, the prescription may be dispensed once only, not later than three months from the date on which it was written.

CHAPTER XIV

THE ACTION OF POISONS, AND THE CIRCUMSTANCES WHICH INFLUENCE IT

Poisons and noxious substances may act—

A. **Locally** on application either to the skin or mucous membranes.
   Corrosives—*e.g.*, strong acids and alkalies.
   Irritants, such as the weaker acids or alkalies, mustard, many mineral poisons, such as arsenic and mercury salts, mechanical bodies, such as powdered glass, etc.

B. **Remotely,** after absorption into the blood, or by shock.
   By shock—*e.g.*, the shock of corrosives.
   By specific action after absorption—
   On brain—narcotics : opium, chloral, etc.
   ,, ,, excitants : alcohol, caffein, etc.
   On cord—strychnine, etc.
   On peripheral nerves—conium, curare, etc.
   On kidneys—cantharides, cubebs, turpentine, etc.
   On salivary glands—mercury, etc.
   On the liver—phosphorus, etc.
   On mucous membranes—arsenic, etc.
   On heart—digitalis, etc.
   On blood-vessels—constringent : ergot, etc.
   ,, ,, dilating : amyl nitrite and other nitrites.

C. **Both locally and remotely**
   Typically oxalic acid, carbolic acid, arsenic, antimony, and other metallic poisons, phosphorus, cantharides, etc., which cause local irritation of the alimentary canal and also produce toxic effects after absorption.

Only a few typical examples have been given under each heading ; a complete list is unnecessary.

Illustrative cases will be found in the part of this work devoted to individual poisons.

Circumstances which Influence the Action of Poisons

A. **Method of Administration.** 1. *By the Mouth.* This is the most frequent method of administration, and must be taken as typical in estimating dose, etc. As a rule, if the dose by the mouth is considered as unity, the rectal dose may be considered to be about one and a half to two, and the hypodermic dose about one-quarter.

The chief toxicological point is that any substance (other than corrosives, irritants, etc.), taken in this way, has to be absorbed through the alimentary mucous membrane, before it produces effects. By the slowness of gastro-intestinal absorption (*vide* p. 235), by alteration during digestion, or by the action of the liver (through which organ many substances passing from the alimentary canal have to pass), some poisons would seem to be rendered almost inert. Snake venom provides an excellent example of this.

The following experiment was made by Taylor: "In June, 1873, Dr. Pavy and I performed two experiments with the dry poison of the cobra di capello, which showed that even in a fasting animal no effects were produced when it was injected into the stomach. Two grains of dry cobra poison were mixed with a small quantity of distilled water and introduced into a wound in the cellular tissue beneath the skin of a rabbit. Symptoms of poisoning showed themselves in a quarter of an hour, and the animal died twenty minutes afterwards. A similar quantity of the cobra poison was injected into the stomach of a healthy young dog which had been kept without food for many hours. No symptoms of poisoning were at any time observed." [1]

2. *Hypodermically.* By this method the substance reaches the general blood stream without passing through the digestive organs. Snake poison, if it is to be effective, can be administered in this way only, at least in small doses. As a rule, the lethal dose is about a third or a quarter of the same drug when given by the mouth, but the exact proportion is very variable. Unless special precautions are taken (*vide* next paragraph), the method is available only for such substances as are soluble in the lymph or tissue juices.

3. *Intravascularly.* The injection of a substance directly into the blood is made use of only in scientific experiments, and for therapeutic purposes, though it is conceivable that it may be unintentionally adopted when the point of the hypodermic syringe by accident just penetrates to the lumen of a vessel. It can only be used for soluble substances unless the results of emboli are to be studied.

4. *Endermically.* Rubbed on to, and absorbed through, the skin, physiologically identical with the hypodermic method. The skin with its horny epithelial covering is a bad absorptive organ, although certain substances (such as mercury, guaiacol, methyl salicylate) appear to pass through it relatively easily, as does mustard gas and other so-called gases used in chemical warfare. Occasionally powders are dusted on to a raw surface for absorption or for local relief of pain, but in these cases the epithelial covering is absent and absorption may then be very free. Poisoning by this means is rarely homicidal, but it may occur from carelessness and accident.

5. *By Rectum, Vagina, or Bladder.* Poisonous substances are occasionally administered by these routes with criminal intent, especially in the procuring of an abortion, but occasionally with homicidal intent. The dose required *per rectum* is usually considered to be about twice that by the mouth, owing to the slowness of absorption from this viscus. The following case was reported from New South Wales:—

A verdict of "Death by misadventure" was returned at an inquest on the body of a woman who died about twenty minutes after an injection of an enema made by steeping about 1¼ oz. of "Yankee Doodle" tobacco in a quart of boiling water.

[1] Guy's Hosp. Rep., 1874, and also many other experiments by the late Sir Thomas Fraser, of Edinburgh.

Six minims of nicotine were extracted from the abdominal viscera. The evidence was conclusive of the nature of the poison and its method of administration, the latter being chosen for the purpose of relieving constipation.

6. *By the Lungs.* A poisonous dose of any drug may be accidentally given when using intralaryngeal injections and applications. Poisoning by way of the lungs is, however, chiefly due to gases such as CO, CO_2, HCN, PH_3, AsH_3, coal-gas, etc. Accidents and suicide with coal-gas are of very common occurrence, and homicide might be thus attempted. Occasionally death takes place from lesions of the lungs when corrosives are taken or when the person is exposed to the fumes of such volatile acids as nitric or hydrochloric, to toxic gases such as ammonia, chlorine, various war gas, benzene, and various hydrocarbons, or to toxic dust (*e.g.*, zinc, mercuric chloride).

Of comparative dosage by the lungs nothing can be said, as the gas may kill by local action, by causing spasm, œdema, pneumonia or broncho-pneumonia, necrosis or hæmorrhage, by its irrespirable nature, or by its action after absorption, as in deaths from chloroform inhalation, carbon monoxide, etc.

The toxic concentrations of these substances vary greatly, but as far as rapidity of action is concerned we may consider the pulmonary route to be in the majority of cases extremely rapid.

B. **Idiosyncrasy.** Of the intolerance exhibited by certain people for certain drugs and even articles of diet there can be no question. Its explanation is extremely difficult and speculative. Fish, eggs, and fruit are common articles of diet which will, even when quite fresh, cause in some people definite toxic symptoms, usually affecting the skin (urticaria), but sometimes of a more general nature such as cyanosis, dyspnœa, asthma, rigors, fever, diarrhœa, hæmorrhage from the bowel and albuminuria. Fatal cases are very rare, but many cases are reported where the symptoms have been serious and apparently dangerous. Potassium iodide and bromide, opium, belladonna and aspirin are common examples of drugs to which many people are allergic.

A fatal case occurred in the London Hospital some years ago in which a child lost its life by the administration of one small dose (one or two grains only) of bromide of potassium. A prominent feature of the illness was a severe bromide rash.

A case is reported where the patient was intolerant to iodoform, grey oil, sublimate, bromides, veronal, morphia, borax, zinc, strawberries and crabs.

It is difficult to understand how advantage of such idiosyncrasy could be taken for homicidal purposes, but accidental cases occur with great frequency. The subject of idiosyncrasy is thus of medico-legal importance when symptoms resembling those of poisoning follow a meal consisting of a particular kind of food. In such a case, without a knowledge of this peculiar condition, we might hastily attribute to poison effects which were really due to another cause.

C. **Age.** This is to some extent connected with idiosyncrasy. There are, for instance, many articles of diet which may justly be called "noxious" or even poisonous for a baby which are wholesome enough for an adult, and *per contra* a healthy boy will eat with impunity many things that might give his father serious cause for regret. Many cases

of criminal neglect in children depend on the unsuitability of the food quite as much as on the lack of quantity, and a medical jurist must be acquainted with the principles of infant feeding. The proportionate dose of a drug for a child is usually reckoned by the following formula, *viz.*: taking x to represent the dose for an adult,

the dose for any age is $= x \times \frac{\text{age in years}}{\text{age in years} + 12}$; thus for a child of six the dose is $\frac{6}{6 + 12} = \frac{1}{3}$ the ordinary dose for an adult. There are also some drugs of which children can take more than their proportionate dose. Mercury and belladonna are two well-known examples. On the other hand, there are some of which they cannot take a proportionate dose. Opium, for example, has to be given very cautiously indeed to young children. The explanation is obscure, but no doubt depends to a great extent on the fact that a child's tissues (in the case of opium probably the cortex of the brain) are very immature, and unusually susceptible to external influences, during the period of growth. Similarly, young persons are more sensitive to carbon monoxide than are adults, a fact connected with the general increased susceptibility of small animals to this poison. It may be recalled that canaries or mice are used—*e.g.*, in coal mines—as indicators of carbon monoxide for this reason.

D. **Habit.** Habit diminishes the effect of certain poisons. Opium, when frequently taken, loses some of its effect after a time, and requires to be administered in increased doses. Confirmed opium-eaters can take, in one dose, a quantity of the drug which would have killed them if they had begun with a dose of the same quantity. Even infants and children, who are well known to be especially susceptible to the effects of opium, and are liable to be poisoned by small doses, may, as a result of habit, become accustomed to taking the drug in large quantities.

According to Grainger, the system of drugging children with opium in the factory districts began soon after birth; and the dose was gradually increased until the child could take from fifteen to twenty drops of laudanum at once. This had the effect of throwing it into a lethargic stupor. Non-habituated children of the same age would be killed by a dose of five drops.

The same effect of habit is to be seen more or less in the use of tobacco, alcohol, ether, cocaine, morphine and other alkaloids. It is usually found in articles derived from the vegetable kingdom, and also with synthetic drugs, such as sulphonal, veronal, chloral, etc. Tolerance for mineral substances is limited, but it occurs in connection with arsenic to a certain extent, as has been reported with reference to the Styrian practice of **arsenic-eating.**

A Styrian took in one day four and a half grains of white arsenic, and on the day following five and a half grains, crushing the mineral between his teeth and swallowing it. The day after he had swallowed the second dose the man left the place in his usual health, and there is no further record of him.

A man once took in the presence of Knapp seven and a half grains of arsenic, which did not produce the slightest visible effect on his feelings. A portion of his urine passed on the same and the following day was found to contain arsenic.

R. C. Maclagan saw one man swallow between four and five grains of arsenic in powder. This man had been accustomed to taking it for a year, having begun with small doses; he did not suffer from any bad effects. Another man, *æt.* 46,

swallowed six grains of arsenic, and washed it down with cold water. Arsenic was detected in the urine about an hour after the poison was swallowed; but as they were habitual arsenic-eaters, it is probable that the eliminated arsenic may have been from that previously taken.

The following case shows that the maintenance of good health in such cases is exceptional.

A man who had carried on the practice of arsenic-eating for three or four years suffered from all the symptoms of arsenical cachexia. He sank under this practice, and after death the usual appearances of chronic poisoning by this substance were found. A chemical analysis showed only slight traces of arsenic in the liver, and none in the stomach.

It is generally agreed that symptoms of acute poisoning are sooner or later the results of the adoption of this dangerous practice in Styria.

The whole question of the effects of arsenic-eating was raised on the occasion of the trial of Mrs. Maybrick,[1] when it was contended that the deceased, Mr. Maybrick, was an habitual taker of arsenic; but the evidence as to this was inconclusive.[2]

Attempts to imitate experimentally the condition in human arsenic-eaters have tended to show that the so-called tolerance to arsenic is probably due to diminished absorption through the intestinal wall. Dogs made "fast" to arsenic *per os* succumbed to $\frac{1}{6\,2}$ of the maximum dose (*per os*) when given subcutaneously (Cloetta).

Habit has generally to be considered in connection with accidental poisoning by an overdose, although it is rarely alleged in homicidal cases. The Maybrick case is the most notorious example of the latter. Evidence of such a habit is usually easy to obtain, and often throws considerable light on the case.

The form in which the question of habit is most frequently raised in medico-legal cases is as follows: whether, while the more prominent effects of a poison are thereby diminished, the insidious or latent effects on the constitution are at the same time counteracted. The answer is of some importance in relation to the subject of life insurance, for the concealment of the practice of opium-eating by a person whose life was insured has already given rise to an action in which medical evidence on this subject was rendered necessary. As a general principle it is true that the habit cannot altogether counteract the insidious effects of poisons, and that the habit is liable to cause disease or to impair health.

E. **Dose.** This is a matter of the greatest importance. Drugs which are constantly being prescribed in small doses would in larger doses prove "noxious" and even poisonous. The medical witness must accordingly be well posted in the official doses of drugs, and especially must he be able to refer to doses which have actually proved fatal.

In general, it may be accepted that the greater the dose of a poison the more unerring and rapid is its effect.

There can be no doubt that there are certain conditions of the system and its organs which appear to confer a power of tolerating large doses of drugs which in other conditions would prove poisonous. Thus persons suffering from tetanus and hydrophobia have taken, without

[1] *R. v. Florence Maybrick*, Notable English Trials. Wm. Hodge & Co.
[2] *Cf. Bostock* v. *Nicholson*, [1904] 1 K.B. 725 (arsenic in beer).

producing dangerous symptoms, doses of opium which in health would have proved fatal and in delirium tremens doses of bromide have been given such as would in health be certainly dangerous. Physiological antagonism (*vide* below, p. 251) may have something to do with the matter, but in many instances the explanation lies in alterations in the rate of absorption and excretion of the drug, and its destruction in the body.

F. **State of Stomach and Kidneys.** These organs have such an important bearing on poisons in general and the effects of varying dosage in particular that rather full attention must be given to them. We have—

1. Disease in general checking the function of absorption from the stomach and excretion from the kidney.

2. Local disease of stomach or kidney causing vomiting or deficient absorption or excretion.

3. Effects of sleep on digestion or excretion.

4. Amount and nature of the contents of the stomach when the poison is taken, influencing action and time of action.

To understand the significance of the point under discussion the following idea must be borne in mind. A dose of non-irritant poison taken into the stomach or injected hypodermically may be divided into three parts, which may be designated x, y, z (x being that part still left unabsorbed in the stomach or intestine or locality of the injection; z that part which has been excreted by kidneys, skin, lungs, or bowel; and y that part which has been absorbed and is still either in the blood or in contact with or within the tissue cells). The symptoms exhibited at any one moment are not due to x nor to z, but almost entirely to y. In corrosives and irritants the symptoms are at first entirely due to x, but later may be, and in many cases are (oxalic acid, phosphorus, etc.), due to both x and y. Now the rate of absorption from the stomach is a function of two variables: (1) the intrinsic power of the substance to pass through the membranes; (2) the condition of the stomach wall. Similarly the rate of excretion from the kidneys (lungs, etc.) is also a function of the same two variables. In health a little information has been obtained with regard to the diffusibility of certain substances which may be thought to exert no specially deleterious effect upon the absorbing or excreting cell in their passage through such cells, but we have no means of knowing how far and in what way poisons may affect these cells. Broadly speaking, the ratio of rate of absorption to rate of excretion gives us a rough measure of y, not one perhaps of great practical value, but certainly one of great theoretical interest. It might, for instance, be raised for the defence of a criminal that the amount of poison found in the stomach (x) or in the tissues (y) was not sufficient to kill. The above consideration shows how such a defence may be met and the amount of bearing it has on the dose administered.

(1) **General Diseases.** There can be no doubt that in acute fevers absorption from the alimentary canal is greatly reduced, which may explain the apparent immunity to certain drugs previously described. Chloral, alcohol and many soluble salts have *per contra* a high specific absorption rate, and the action of large doses of these, as administered in delirium tremens, touches on the ground of physiological antagonism which will be mentioned later (pp. 251 *et seq.*).

On the other hand, a person very feeble from disease may succumb to a dose that is ordinarily safe. Antimony, for example, might thus kill by syncope, as it is a powerful depressant. Opium, again, should be avoided in conditions of coma. A knowledge of these facts is of importance in reference to charges of malpraxis when death has arisen from ordinary or extraordinary doses of medicines administered to persons suffering from disease. In such cases another mode of treatment should be substituted, or a smaller dose than usual given, and its effects carefully watched.

For the absorption of poison administered *per os*, not only is the condition of the mucous membrane of the gastro-intestinal tract of the utmost importance, but also the site where the absorption takes place. For example, with a normal mucous membrane bismuth subnitrate is not absorbed at all, and large doses may be administered without ill effect, but in diseased conditions absorption and intoxication may ensue.

It is not quite so clear that excretion from an otherwise healthy kidney is similarly affected by pyrexial disease. The diminished excretion of urine in fevers may be due to increased excretion from the lungs, though it may be due to the effects of toxins in the blood on the secreting cells of the kidney.

(2) **Local Gastric Diseases.** Inflammatory conditions of the stomach may increase the rate of absorption of certain drugs, but on the other hand the irritability is increased and may lead to excessive vomiting and loss of a great part of the drug. In pyloric stenosis and conditions of atrophy and ulceration from cancer there can be no question but that absorption is interfered with.

The similarity between the symptoms of chronic irritant poisoning and gastric disease has probably caused many cases of crime to be overlooked, discovery only coming either by accident or on account of a suspicious frequency in the manner of death (*e.g.*, Palmer's case or Chapman's case) : in fact almost all notorious criminal cases of poisons have been concerned with arsenic or antimony as the means used when slow poisoning has been in question.

(3) **Local Disease of the Kidney.** The rate of excretion of a drug is naturally affected by any diseased state of the kidney and there is a tendency for cumulative action after the administration of normal doses of many drugs when local disease is present. Similarly the toxic effect of poisonous doses is likely to be greatly enhanced. When giving drugs known to be slowly excreted, constant examination of the urine may give an early warning of insufficient elimination. The condition of the kidneys may be of importance in any case of poisoning, for a dose which might have little effect on a healthy person might kill one whose kidneys were diseased.

(4) **Effects of Sleep.** The slowness of absorption from the stomach during sleep has to be remembered in cases in which the symptoms of poisoning have been delayed, and when it is found that for part of or all the time the victim has been asleep.

(5) **Amount and Nature of the Contents of the Stomach.** The chemical interchanges between the substances which are placed in the stomach do not go on in quite the same manner as they do in a test tube, though

they may be approximately the same. Food, however, prevents *contact* with the stomach wall to some extent, and thus diminishes the effects of some less soluble irritants; at the same time *dilution* has to be considered when the stomach contains much liquid or bulky pultaceous contents; *absorption* is also influenced in some physiological manner by the presence of food and by its preparedness for absorption, and certain poisons react chemically with constituents of food or partially digested foods (*e.g.*, corrosive sublimate forms insoluble compounds with proteins). Arsenic, acids, alkalies, and some other irritants are typical examples of poisons the action of which is hindered and delayed by the presence of food in the stomach. *Per contra*, when the stomach is empty, more rapid and intimate contact with the mucous membrane, less dilution and quicker absorption are all favoured; it is easy to see the bearing of these factors on the question of the dose of a poison in comparison with the rapidity of its action.

G. **Concentration.** This chiefly affects irritant poisons which, when concentrated, become almost corrosive in their action. The acids and alkalies, for instance, when concentrated, will burn the mouth, gullet, and stomach, and a dose which might lead to a fatal issue may in weaker dilution be swallowed with impunity; of drugs which act after absorption, concentration has *per se* no consequence; it is the concentration in the blood or tissues after absorption which is of importance.

H. **Chemical Combination.** The toxic effect of elements may vary greatly with variations in their chemical combinations. In simple salts this may be largely due to the difference in solubility and in the readiness whereby they are absorbed into the cells of the body. Such differences are seen for example in the two chlorides of mercury or the oxides and sulphate of arsenic. In the more complex substances, a different arrangement of the atoms within the molecules may cause completely different effects from apparently similar substances.

I. **Physical State** (gas, liquid or solid). The portal of entrance and the method of administration are important factors which must be considered along with the physical state of the poison. In general, gases produce their effects with great rapidity when breathed into the lungs. When taken by the mouth liquids act more quickly than solids, fine powders more rapidly than coarse powders and of course the solubility of the powder in the gastric fluid conditions its absorption.

Perhaps the fact may be mentioned here that hard old pills will often enough pass unchanged through the alimentary canal. Keratin, again, is a substance (insoluble in the acid gastric juice, soluble in the alkaline contents of the small intestine) used to coat pills intended to become active only in the intestine; a poison given in a keratin coat might have its action delayed several hours. *Vide* also *ante*, p. 221, where a berry of cocculus indicus still in its husk was very justly termed a poison.

These considerations on the circumstances influencing the action of poisons may appear a little prolix and commonplace, but they are very germane to the subject, for they enable the medical jurist to see

the drift of, and to answer so far as explanation is possible, the following questions which a defending counsel may ask :

What was the actual cause of death ?
Why was it due to poison and not to disease ?
What is the fatal dose of the alleged poison ?
Why did the symptoms not occur at once, or why did they ?
Why did they vary from the usual time of occurrence ?
Was the dose necessarily a fatal one ?

Many other similar questions will probably be asked by a defending counsel in his endeavours to show that death was not caused by poison at all, or if so, that it was not the alleged poison, because (*a*) the symptoms did not occur at the usual time, or (*b*) in the usual order.

The Time at which Symptoms appear after swallowing a Poison. This question requires examination, because the more common poisons, when taken in fatal doses, generally cause death within definite periods of time. By an attention to this point we may, in some instances, be enabled to negative a charge of poisoning, and in others to form an opinion of the kind of poison which has been taken. In a court of law a medical practitioner is often required to state the usual period of time within which poisons prove fatal. Not only do poisons differ from each other in this respect, but the same substance, according to the circumstances in which it has been taken, may differ in the rapidity of its action. A large dose of prussic acid, *i.e.*, from half a fluid ounce to an ounce of 2 per cent. acid, may cause death in less than two minutes. In ordinary cases of poisoning by this substance a person dies—*i.e.*, all signs of life will usually have ceased—in from ten to twenty minutes ; if he survives half a hour, there is some hope of recovery.

In the case of seven epileptics, accidentally poisoned by a similar dose of this acid in one of the Parisian hospitals, the first died in about twenty minutes, the seventh survived three-quarters of an hour.

Oxalic acid, one of the most active of the common poisons, when taken in a dose of from half an ounce to an ounce may cause death in from ten minutes to an hour : if the poison be not perfectly dissolved when swallowed, it is slower in its action.

The strong mineral acids, in fatal cases, cause death in about eighteen or twenty-four hours.

Arsenic in the form of arsenious oxide (white arsenic) operates fatally in from eighteen hours to three or four days. But in more than one case death was caused within two hours.

In *R.* v. *Russell* a woman was convicted of the murder of her husband by poisoning him with arsenic. The poison was detected in the stomach ; but the poisoning was disputed by some medical witnesses because (among other reasons) the deceased had died *three* hours after the only meal at which the poison could have been administered to him.

The authority of Sir A. Cooper and others was cited to show that, according to their experience, they had never known a case of poisoning by arsenic to have proved fatal in less than seven hours. This may be admitted, but, at the same time, there was sufficient authority on the other side to establish that some cases had actually proved fatal

in three or four hours. So far as this objection was concerned, the prisoner was properly convicted. With reference to the medical question raised at this trial two cases have occurred in which the patients died within *two hours* after taking arsenic; and several cases have been reported in which death has taken place in from three to four hours after the administration of this poison. It is quite obvious that there is nothing, so far as we know, to prevent arsenic from causing death in an hour, or even within a shorter period. These matters can be settled only by a careful observation of numerous cases, and not by any *à priori* reasoning, nor by a limited individual experience.

Opium, either as a solid or in the form of laudanum, commonly proves fatal in from six to twelve hours; but it has been known, in several instances, to cause death in less than three hours; on the other hand, it has never been known to cause death instantaneously or within a few minutes; those who survive the effects of this poison for twelve hours are considered to have a fair chance of recovery. This must be understood to be merely a statement of the average results, as nearly as we are warranted in giving an opinion; but the medical witness will of course be aware that the fatal period may be protracted or shortened, according to the circumstances above detailed.

In cases of **sudden death** there is often a tendency on the part of the public to suspect poisoning. They do not consider that persons may die a natural death *suddenly* or, as we shall presently see, that death may really take place slowly, and yet be due to poison. This prejudice often gives rise to the most unfounded suspicions of poisoning, and, at the same time, leads to cases of chronic or slow poisoning being mistaken for natural disease.

Chronic Poisoning. When life is destroyed rapidly by poison, the case is said to be acute, to distinguish it from *chronic* poisoning in which death takes place slowly. Chronic poisoning is a subject which frequently requires medico-legal investigation. Most poisons, when their effects are not rapidly manifested, owing either to the smallness of the dose or to timely treatment, are capable of slowly undermining the powers of life and killing the patient by producing emaciation and exhaustion. This is sometimes observed in the action of arsenic, corrosive sublimate, and tartarated antimony.

When only *traces* of arsenic, antimony, phosphorus and a few other minerals are found, defending counsel may raise the question whether they are not natural constituents of the body or of certain articles of food: the discovery of arsenic in oysters, prawns, and certain fish,[1] and the epidemic of poisoning from the presence of arsenic in beer at Manchester and elsewhere, have shown how many hitherto unsuspected sources of this element, and unsuspected channels of its admission to the body, there may be.

Several cases have come before the courts in which the facts connected with chronic poisoning were of some importance; in most cases murderers kill their victims by administering poison in large doses; but in the cases referred to, small doses were given at intervals, a fact which, in some of them, led to a doubt of the real cause of the symptoms.

[1] Chapman, *The Analyst*, November, 1926.

In *R.* v. *Winslow* the prisoner was charged with the murder of a woman by administering small doses of antimony. The suspicion of her physician was aroused by the intermittent and violent nature of the vomiting, as well as by the extreme depression. Antimony was found in the urine and fæces; and, after death, this substance was discovered, in small quantities, in the viscera. The deceased was at the time suffering from malignant disease of the cæcum, but it was alleged that the antimony had accelerated her death. The jury acquitted the accused. The examination of the bodies of the sister of deceased, and of two other members of the family, led to the discovery of antimony, also in small quantity, in the viscera of each; and from the nature of the symptoms preceding death, as well as the general healthiness of the organs, no doubt was entertained by the medical witnesses that all these persons, members of the same household, had died from the effects of the antimony administered at intervals in small doses.

A set of cases somewhat similar came to light by inquests on exhumed bodies at Bilston. Three children in a family died at different times, under similar symptoms. Antimony was found in two of the bodies, and the body of a third child was exhumed after two months' burial, and antimony was also found in it. This child died on October 10th, and its death was registered on the 13th of that month as death from "asthenia," and "gastric fever" "six days." They all received medical attendance, and their names were entered in a burial club.

In Chapman's case (or Klosowski), the victim had been a patient of Guy's Hospital for what was probably illness due to antimonial poisoning without discovery of the crime; the murderer's previous victims had been buried on ordinary certificates.

The case is given in full on pp. 275 *et seq.*

In the notorious case of William Palmer, one physician called for the defence affirmed that the symptoms under which Cook died were those of angina pectoris; and another physician, called for the defence, attributed death to epilepsy with tetanic complications. With reference to the death of Ann Palmer, which was caused by repeated doses of antimony, the solid sulphide of this metal was found in the stomach after death, and the metal itself pervaded the whole of the tissues. A respectable physician, with only a superficial knowledge of the facts of the case, wrote a pamphlet to prove that this woman had died from an attack of cholera. If these persons had been called in to attend these two victims of secret poisoning while living, it is quite obvious that they would have had no suspicion of poisoning, and that they would have respectively certified that death was caused in the one case by angina pectoris or epilepsy, and in the other by cholera. They would thus have effectually screened, under erroneous medical certificates, the acts of a man who was possibly the greatest criminal of his age. If physicians of some standing, and professed experts, can thus fail to detect ordinary cases of poisoning, the possibility of error in granting medical certificates in cases of poisoning is obviously very real.

It is much to the credit of the medical profession that the crime of murder by poisoning—a form of death from which no ordinary caution or foresight can protect a person—is so frequently brought to light by the announcement of suspicious facts of a medical nature made to magistrates and coroners; and on several occasions the highest compliments have been paid by judges to medical practitioners who have been thus indirectly the means of bringing atrocious criminals to the bar of justice.

CHAPTER XV

DIAGNOSIS OF POISONING

We now proceed to consider the evidence of poisoning in the *living* subject. To the medical practitioner the diagnosis of a case of poisoning is of great importance, for by mistaking the symptoms produced by a poison for those arising from natural disease, he may omit to employ appropriate remedial measures with the result that the patient loses his life. To a medical jurist, a correct knowledge of the symptoms furnishes the chief evidence of poisoning in those cases in which persons are charged with the malicious and unlawful administration of poison. The symptoms produced during life constitute also an important part of the evidence in cases where poison results in death.

Poisoning, like other forms of violence of which the law takes notice, occurs either Accidentally, Suicidally, or Homicidally.

So far as diagnosis is concerned, the first two classes are not likely to give rise to so much trouble as the third, for conclusive evidence as to the nature of the case is usually obtained readily. There are, however, many diseases with incidents in their course which may closely resemble poisoning, and a murderer may endeavour to make the case resemble one of natural illness or of suicide.

Speaking broadly, there is no *single symptom*, and no definite group of symptoms, which is absolutely characteristic of poisoning. On the other hand, there is no poison which produces the finer shades of difference from day to day and the changing aspects of disease, and no disease which gives all the features of poisoning in number and in sequence. The closest resemblance to disease is produced by food poisoning, and the closest resemblance to poisoning is produced, perhaps, by uræmia in its acute phases ; but except for the associated circumstances of the means by which the poison is introduced into the body these are really illustrations of identicals, and they scarcely invalidate the above generalisation.

The following are some of the principal conditions which should arouse suspicion of poisoning and lead one to make particular search for their causation :

1. **In Acute Poisoning, the Symptoms appear suddenly.** It is the common character of most poisons, when taken in the large doses in which they are usually administered with criminal intent, to produce serious symptoms, either immediately or within a very short period after they have been swallowed.

This suddenness of onset is most marked when corrosives and irritants (concentrated or in large doses) are in question, for they have an immediate local effect of very severe nature. Less powerful irritants

act more slowly, although the onset of vomiting is usually sudden. Poisons which act only after absorption produce their symptoms more gradually in proportion to the slowness of absorption, which we have discussed above.

In this connection the onset of the " acute abdomen " of the surgeons must not be forgotten. Sudden acute pain followed by vomiting are the prominent features of the surgical, as they are of the toxicological problem. The differential diagnosis is not usually difficult in practice. When, however, small repeated doses of a poison are given, the absorption may be slow and lead to a gradual development of symptoms resembling disease.

2. **The Symptoms appear during a State of Health.** Symptoms of acute poisoning manifest themselves in a person while in a state of *health*, without any apparent cause. This rule is open to numerous exceptions, because the person on whose life an attempt has been made may be actually suffering from disease ; and in these circumstances the symptoms may be so obscure as to disarm all suspicion. When poison is secretly given in medicine, a practitioner is very liable to be deceived, especially when the disease from which the person is suffering is of an acute nature, and has been attended by symptoms of disorder in the alimentary canal. Several cases of poisoning have occurred in which arsenic was criminally substituted for or mixed with medicine, and given to persons whilst labouring under a disorder of the bowels. We are justified in saying, however, with respect to this character of poisoning, that when, in a previously healthy person, violent vomiting and purging occur suddenly and without any assignable cause (such as pregnancy, disease, or indiscretion in diet) to account for them, there is strong reason to suspect that an irritant poison has been taken. When the person is already suffering from disease, we must be especially watchful on the occurrence of any sudden change in the nature or violence of the symptoms, unless such change can be accounted for on common or well-known medical principles. In most cases of criminal poisoning we meet with alarming symptoms without any obvious or sufficient natural causes to explain them. The practitioner will of course be aware that there are certain diseases which are liable to occur suddenly in healthy people, the exact cause of which may not at first sight be apparent ; strangulation of a hernia, perforation of a gastric ulcer, and cerebral hæmorrhage may be taken as typical illustrations ; therefore this criterion is only one out of many on which a medical opinion should be founded.

3. **In Poisoning the Symptoms appear soon after a Meal, or soon after some kind of Food or Medicine has been taken.** This is the most important character of poisoning in the living subject. It has been already stated that most poisons begin to operate within about an hour after they have been swallowed ; and although there are a few exceptions to this remark, yet they occur in circumstances easily to be appreciated by a practitioner. Thus, if the symptoms observed in the patient are due to poison, the substance has most probably been swallowed, either in food or medicine, from half an hour to an hour previously. It must be observed, however, that cases may occur in which the poison has not been introduced by the mouth (*vide* above, " Method of Administration "). Oil of vitriol and other corrosive liquids have been injected

into the rectum, with a fatal result ; the external application of arsenic, corrosive sublimate, and cantharides to ulcerated surfaces has destroyed life. In one case in which arsenic was introduced into the vagina, the woman died in five days with all the symptoms of arsenical poisoning. Such cases are rare, but, nevertheless, the certainty that they have occurred, where their appearance could hardly have been anticipated, shows that in a suspicious case a practitioner should not deny the fact of poisoning merely because it may be proved that the person could not have taken poison in the usual way, by the mouth. Again, persons may be destroyed by the vapours of coal-gas, ether, chloroform, prussic acid, or other powerful volatile poisons, introduced into the body through the lungs. Such a mode of suicide, or murder, might disarm suspicion, from the fact that no noxious material was found in the stomach.

Let us suppose, however, the circumstances to have been such that these secret means of destruction could not have been resorted to, and that the substance is one of those most commonly selected by a murderer, such as arsenic, tartar emetic, oxalic acid, or corrosive sublimate, then we may expect that this character of poisoning will be made evident to us, and that something must have been *swallowed* by the patient shortly before the alarming symptoms appeared. By observations attentively made, it may be in our power to connect the appearance of the symptoms with the use of a particular article of food, and thus indirectly lead to the detection of a criminal. Supposing that many hours have passed since food or medicine was taken by the patient, without any effect ensuing—it is probable that the symptoms are due to natural causes, and not to poison.

When symptoms resembling those of poisoning speedily follow the ingestion of food or medicine, there is reasonable ground for suspicion ; but caution should be observed in drawing inferences, since the most extraordinary coincidences sometimes present themselves.

Delay in the onset of symptoms occasionally occurs in phosphorus poisoning if the dose is small, in food poisoning, and in poisoning with certain organic substances.

The substitution of poisonous mixtures for medicinal draughts or powders is by no means unknown, although it might be supposed to indicate a degree of refinement and knowledge not commonly to be found among criminals. Medical practitioners are thus liable to be imposed upon, and the following case should serve as a caution.

An apothecary prepared a draught, into which another person put poison, intending thereby to kill the patient for whom the medicine was prescribed. The patient, not liking the taste of the draught, and thinking there was something suspicious about it, sent it back to the apothecary, who, knowing the ingredients of which he had composed it, and wishing to prove to his patient that he had done nothing wrong, drank it himself, and died from the effects. He was thus the unconscious agent of his own death ; and though the draught was intended for another, the party who poisoned it was held guilty of murder.

On the other hand, the occurrence of symptoms resembling those produced by poisoning, soon after food or medicine has been taken, may be a pure coincidence. In such a case, poison may be suspected, and it will be the duty of a medical jurist to guard against the encouragement of such a suspicion, until he has strong grounds to believe it to be well founded. No public retraction or apology can ever make amends

for the injury which may in this way be inflicted on the reputation of another; for those who hear the accusation may never hear the defence. In all such cases, a practitioner may entertain a suspicion, but until confirmed by facts, he should avoid *expressing* it publicly. When death is not a consequence, it is difficult to clear up such cases, except by the aid of a chemical analysis; but this, as we know, is not always available or possible. If death ensues, the real cause is usually apparent, and a suspicion of poisoning is thus often removed by an examination of the body.

In cases of chronic criminal poisoning suspicion is often first aroused by the *repetition* of such an incident as the above. Many patients may be sick once after a dose or a meal, but this excites probably no suspicion, nor need it do so. If, however, they are repeatedly sick, we may with justice suspect criminal intent.

4. **In Poisoning, when several partake at the same time of the same Food or Medicine (mixed with Poison), all suffer at approximately the same time from approximately the same Symptoms.** This feature of poisoning cannot always be observed; but it furnishes good evidence of the fact when it exists. Thus, supposing that after a meal taken by several persons from the same dish only one suffers, the suspicion of poisoning is considerably weakened. The poisoned article of food may be detected by observing whether they who suffer from symptoms of poisoning have partaken of one particular solid or liquid in common.

In a case of accidental poisoning at a dinner party, a medical man who was present observed that those who suffered had taken port-wine only; the contents of the bottle were examined, and were found to be a saturated solution of arsenic in wine.

As a rule, considerable reliance may be placed upon this feature, because it is improbable that any disease should suddenly attack many healthy persons at the same time, and within a short period after having partaken of food together. We must beware of assuming that, when poison is in fact present, all will be attacked with precisely similar symptoms, because there are many circumstances which may modify their nature and progress. As a rule, that person who has partaken most freely of the poisoned dish will suffer most severely; but even this does not always follow. In a well-known case of the several persons who partook of a dish poisoned with arsenic, those who had eaten little and *did not vomit* speedily died; whereas others who had partaken largely of the dish, and had in consequence vomited freely, recovered, since the retention in the stomach of the poison meant an increase in the amount absorbed, whereas the free vomiting removed the bulk of the poison.

When conducting inquiries it should be remembered that symptoms resembling those produced by irritant poison may sometimes be traced to *food*. Meat rendered unwholesome by disease or decay, pork, bacon, sausages, cheese and bread, as well as certain kinds of shell-fish, may give rise to symptoms of poisoning, and even cause death. Such cases may be regarded as poisoning by bacterial infection or by animal or vegetable toxins. All the characters above described as indicative of poisoning may be observed, and the difficulty of forming an opinion is often increased by the fact that some of the persons attacked may

have previously partaken of the same kind of food without inconvenience. Instances of this form of poisoning are common (*vide infra*, "Food Poisoning").

The statement that there is no disease resembling poisoning which is likely to attack several healthy persons at the same time, and in the same manner, is undoubtedly true as a general principle, but the following case will show that mistakes may occasionally arise even in these circumstances. It occurred in London during prevalence of malignant cholera early in the nineteenth century.

Four of the members of a family, living in a state of great domestic unhappiness, sat down to dinner, apparently in good health; some time after the meal, the father, mother, and daughter, were suddenly seized with violent vomiting and purging. The evacuations were tinged with blood, while the blueness of the skin, observable in cases of malignant cholera, was absent. Two of these persons died. The son, who was known to have borne ill-will against his father and mother, and who suffered no symptoms on this occasion, was accused of having poisoned them. At the inquest, however, it was clearly shown by the medical attendant that the deceased persons had died of malignant cholera, and that there was no reason to suspect that any poison had been administered.

In this case symptoms resembling those of irritant poisoning appeared suddenly in several persons in perfect health, and shortly after a meal. The utility of any rules for investigating cases of poisoning depends entirely on the judgment and discretion with which they are applied to particular cases.

5. **Course of the Symptoms.** In practically all accidental and suicidal cases, the course of the symptoms is either steadily downhill to death or equally steadily uphill to recovery; phosphorus is the principal exception that is not obvious at first sight; and the after-effects of corrosives and a few of the mixed class that act locally and remotely must also be remembered.

In cunning homicidal poisoning there may be ups and downs in the symptoms of the victim, but these from their nature will arouse suspicion. Some diseases have a somewhat similar course, and especially those, such as acute gastritis, which may at first sight be mistaken for poisoning. The point is, however, to be remembered in suspicious cases.

The average course of the disease and the expected action of administered medicine are known, and it is from discrepancies between the expected and the realised that suspicion arises.

6. **The Discovery of Poison in the Food taken, in the Matters vomited, or in the Excretions.** One of the strongest proofs of poisoning in the living subject is the detection of poison either in the food taken by the person, or in the matters vomited, or, after the lapse of a few hours, in the urine. The evidence is more satisfactory when the poison is detected in the vomit, and more especially in the urine (*vide infra*), than when it is detected in the food; because this will show that it has really been taken. If the vomited matters have been thrown away, the remains of the suspected food should be examined. Should the results in both cases be negative, and no trace of poison be found in the urine, it is probable that the symptoms were due to disease.

In the investigation of a case of poisoning in a living subject, a medical jurist must remember that poisoning is sometimes *feigned*, and sometimes *imputed*. It is easy for an artful person to put poison into food, as

well as to introduce it into the matters vomited or discharged from the bowels, and to accuse another of having administered it. There are few of these accusers who go so far as to swallow poison in such circumstances, as there is a great dread of poisonous substances among criminals; and it will be at once apparent that it would require a person well versed in toxicology to feign a series of symptoms which would impose upon a practitioner at all acquainted with the subject. In short, the difficulty reduces itself to this :—What inference can be drawn from a chemical detection of poison in food? All that a medical man can say is, "poison is or is not present in a particular article of food": he must leave it to the authorities of the law to develop the alleged attempt at administration. If the poison has been actually administered or taken, then we should expect to find that the person had suffered from the usual symptoms. The absence of these symptoms would be a strong fact against the alleged administration. The detection of poison in the matters vomited affords no decisive proof that it has been swallowed, except under two circumstances. First—When the accuser has actually suffered from the usual symptoms of poisoning, in which case there can be no feigning, and the question of imputation is a matter to be established by general evidence. Secondly—When the matters are actually vomited into a *clean vessel* in the presence of the medical attendant himself, or of some person on whose testimony perfect reliance can be placed. The detection of absorbed poison in the *urine* or *saliva* furnishes a clear proof that poison has been taken, that it has passed into the blood, and has been subsequently eliminated by the kidneys or the salivary glands.

When poison is discovered mixed with food in the stomach it does not necessarily follow that it has been administered in that particular article of food. Should the person have partaken of liquid food, such as milk or gruel, subsequently to the swallowing of poison, these fluids will necessarily acquire a poisonous impregnation from that already contained in the stomach. The patient may have taken the poison in one kind of food, when another and an innocent description of food might thus inadvertently be pronounced to have been the vehicle.

7. **No Poison Found.** Throughout the section dealing with the particular poisons there will frequently be observed cases in which no trace of poison has been found on analysis, although from other circumstances it was almost or quite certain that poison was the cause of death or illness; it is therefore necessary that the position should be examined.

There are three possible explanations of such a result :—

1. *The whole case may have been misunderstood.* We have already drawn attention to the occasional resemblance between the effects of poison and disease, and the fact that no poison has been found must be allowed due weight in balancing the probabilities of the case.

2. *The poison may have been eliminated by vomiting or other means, or neutralised.* In corrosive and irritant poisons a considerable proportion of the poison is lost by vomiting and diarrhœa. It is necessary therefore to save the whole of the vomited matter and other discharges for analysis. The local changes in the stomach, in the shape of inflammation, etc., will be strong corroboration (or negation). In considering the other channels of elimination it must not be forgotten that a poison

may start processes which may continue after the poison has ceased to act, and hence a person may die some time after the poison was administered, from what is apparently disease, a gastritis, for instance, or a nephritis; under the headings of antimony, arsenic, etc., illustrative cases will be found.

3. *The analysis may have been faultily performed.* This may be due to circumstances over which the analyst had no control, *e.g.*, the wrong material sent for analysis, etc., or to circumstances over which he would have had control had he been more skilled. There are a number of substances, which may be fatal to a human being if they gain access to his body, which are beyond the reach of chemical detection and identification, such as the toxins of some bacteria, and there are a certain number of vegetable products which cannot be definitely recognised by analytical methods.

In estimating dosage and quantities, for use in medico-legal cases, it is important, in reference to the presence of absorbed poison in the *tissues*, to observe that it may be found in them at an early period, when it is either absent or only doubtfully present in other parts, such as the alimentary canal.

A man died within *four hours* after he had been attacked with symptoms of poisoning by arsenic. Arsenic was found in small quantity in the stomach, duodenum, and rectum. It was also detected in the liver and spleen; and the proportion found was greater in the latter than in the former organ.

A man died from the effects of arsenic in the most acute form, soon after his admission into Guy's Hospital. He had swallowed a large dose of the poison in water. He was brought to the hospital, and died soon afterwards. Barely *three hours* could have elapsed from the time at which the poison was taken until his death. There were the usual appearances in the stomach, and gritty portions of arsenic with coagulated masses of mucus and false membrane were found in the contents. The intestines were inflamed, and portions of arsenic were discovered as low as the cæcum. Arsenic was found abundantly in the stomach, and a comparatively large quantity of the poison was detected in half an ounce of the dried liver, as well as in the spleen and kidney.

Hence it is obvious that the poison may be rapidly absorbed and copiously deposited within *three* hours, the quantity thus found depending apparently on the dose taken and on the nature of the poison.

Most organic poisons are rapidly destroyed in, or eliminated from, the body but metallic poisons, on the other hand, may be retained for long periods, for example in the keratin tissues such as the hair, nails, and skin in the case of arsenic, and in the bones, especially in the case of lead. Storage in the liver and kidneys is as a rule of short duration.

CHAPTER XVI

WHAT TO DO IN CASES OF POISONING

The duties of a medical practitioner in cases of poisoning are twofold. He has first to undertake the care and treatment of the patient, and secondly to do his duty as a citizen in helping the authorities in arriving at a correct interpretation of the case.

When treating, or called upon for the first time to treat, a case of illness, a medical man's mind must be in one of three conditions :

(*a*) A state of ignorance, one totally unaware of the necessity of thinking of poison.

(*b*) A state of certainty that it is a case of poison or of disease.

(*c*) A state of suspicion that it may be poison.

(*a*) The state of **ignorance** will commonly be dispelled by rumour or gossip, or perhaps more suddenly by a coroner's order for a *post-mortem* examination or an exhumation order from the Home Secretary. It need not be further discussed here, except to emphasise the advice to all medical men to keep in writing as full notes of all their cases as time and circumstances will permit, so that when faced with one of the above crises they may have at least a good basis to work upon.

(*b*) The state of **certainty** of disease has no interest for us here, and the certainty of poison will be considered later.

(*c*) The state of **suspicion** is one of very great delicacy and difficulty. There are three things which should be done at once. First, some of the urine or vomit, or of both, should be submitted to analysis. It is better to have this analysis carried out by an analytical chemist rather than by oneself, because corroborative evidence from an independent witness is thus obtained in one direction or the other. It is easy for a medical man to obtain such specimens as a rule, because they are looked upon as things he would naturally wish to examine for clinical purposes. Secondly, it is advisable to have a consultation with a practitioner of standing, and submit to him his suspicions under seal of professional secrecy, and discuss the matter thoroughly. Thirdly, he should have the patient removed at once to a hospital or nursing home, or if that cannot be done and if circumstances permit, two reliable nurses should be engaged, one for day and one for night duty, who should administer themselves everything in the shape of medicine and food and allow no one to be with the patient alone. The nurses should be of the doctor's own choosing, for it is obvious that the more he can tell them of the exact observations he requires, the better and more loyal assistance will they be able to render him.

The practitioner's own observations, coupled with the information he will have obtained from the above procedure, will nearly certainly bring

suspicions to a head in one direction or the other. If he finds no corroboration of them he is bound to dismiss them for the time and wait for further developments; if on the other hand he obtains such corroboration as converts suspicion into certainty, a very delicate position may, or rather will, arise. "Shall I allow the patient to stop where he is?" is the first obvious dilemma; to remove him is certainly the best possible thing to do, or to banish the guilty party (if found). But either course may be impracticable; so the next question must be, "To whom shall I speak?" He *must* take steps to protect his patient, and to prevent further administration of poison, and he has the choice of informing:

1. **The Victim himself.** If he is an adult and possesses a good deal of strength of will and retains his mental faculties, it is easy to imagine circumstances in which this is the best course to pursue, for the doctor may possibly get from the patient power to act vigorously. If the victim is a child or a person permanently or temporarily *non compos mentis*, it is obvious that telling the victim is useless.

2. **A Member or Members of the Family.** This, of course, depends entirely upon who they are, their peculiar characteristics, their strength of character, etc.

3. **The Police.** Here one must always remember that a doctor is or should be the confidential friend of his patients, at least so far as professional matters are concerned, and police and detective work forms no proper part of his duties. Every case of poisoning is not necessarily criminal; it may be accidental, a possibility which must be excluded before taking such a positive step as informing the police, at any rate in non-fatal cases—of course in fatal ones the coroner *must* be informed at once. On the other hand, such cases usually are crimes, and in bringing such to the notice of the police there is no need to consider the feelings of the perpetrators. At the same time it is necessary to be sure that there are good grounds for the accusation, otherwise a medical man may find himself involved in a trial for libel or defamation of character. Taylor remarked on this subject in former editions of this work:

> When a practitioner is aware of a case of *poisoning*, it is necessary that he should know to what points he ought to give his attention. Every effort should be made by him to save life when the individual is living; but while engaged in one duty, it is also in his power to perform another, supposing the case to be one of *suspected* criminal poisoning, namely, to note down many circumstances which may tend to detect the perpetrator of a crime. There is no person so well fitted to observe these points as a medical man; but it unfortunately happens that many facts, important as evidence, are often overlooked. The necessity for observing and recording them is not perhaps generally known. A medical man need not make himself officious on such occasions, but he would be unmindful of his duty as a member of society if he did not aid the cause of justice by extending his scientific knowledge to the detection of crime.

In non-fatal cases of suicidal poisoning the doctor's position is an extremely delicate one. On the one hand, he may be placed in the position of compounding a felony if he holds his tongue; on the other hand, by reporting the case he may raise such a scandal as may mean ruin not only to himself, but to his patient and his family. There can be no doubt that the correct attitude is to report the matter; but probably many such cases are hushed up without material harm to the community.

By s. 17 of the Food and Drugs Act, 1938, if a medical practitioner becomes aware or suspects that a patient whom he is attending within the district of a Local Authority is suffering from food poisoning, he must immediately send a certificate to the Medical Officer of Health, and must also state whether the case has occurred in his private practice, or in his practice as a medical officer of a public body or institution.

Treatment of Cases of Poisoning

When called to an undoubted case of poisoning, immediate treatment must be the first and only concern. Only the general principles of treatment will be dealt with here, but the practitioner is advised to carry a handy book on treatment in his antidote bag.

The obvious points are—

1. **Empty the Stomach.** This applies to all cases where the poison has been taken by the mouth. The means for doing it are—

(*a*) By the stomach-tube. This is contra-indicated in corrosive poisoning owing to the danger of tearing, and should be used with great caution in irritants.	(*a*) Be careful to introduce water into the stomach before syphoning out the contents, so that the tube may not come into too close contact with the walls, and also that the water may act mechanically in bringing away the "noxious substance" as well as by dissolving it.
(*b*) Emetics :	
Sulphate of zinc.	Thirty grains may be given at once and repeated in a quarter of an hour. It is not very depressing, but if emesis does not occur is apt to be irritating and may have unpleasant after-effects.
Ipecacuanha.	Twenty to thirty grains of the powder or from two to six drachms of the wine, and repeat. Is somewhat depressing, but has no specially deleterious effect.
Mustard and water.	A tablespoonful of the powder may be stirred in a tumbler of water and swallowed rapidly.
Salt and water.	Also excellent used like mustard.
Ammonium carbonate.	Not so efficacious, but is a stimulating emetic; 15 to 30 grains may be given.
Apomorphine.	Given by hypodermic injection, and so may be used when swallowing is impossible. Its drawback is that it is very depressing. Dose : one-tenth of a grain repeated in half an hour if necessary.

2. **Neutralise what remains in the Stomach.** This neutralisation may be of either a direct chemical nature or of a mechanical or physico-chemical nature. The commoner illustrations are—

Alkalies neutralise acids by direct chemical action. In this illustration there is a caution to be given, *viz.*, it is safer to give a little weak solution of hydroxide (Am, Ca, Na, Mg, K), than the respective bi-carbonates or carbonates, because of the possible risk of rupturing the stomach with the liberated carbonic acid. Scrapings from a white-washed ceiling or wall can be used if no other alkali is available (*vide* "Oxalic Acid Poisoning," p. 378).

Acids neutralise alkalies by direct chemical action. Vinegar is most easily obtained and is the safest acid to administer.

Common salt (NaCl) decomposes silver nitrate by direct chemical action, forming the insoluble silver chloride, but this should be got rid of as soon as possible, for it is distinctly a "noxious substance."

Albumen precipitates $HgCl_2$. The action is partly physical and partly chemical, and hence white of egg is the best antidote in corrosive sublimate poisoning. The resultant precipitate, however, must be got rid of as soon as possible, for it again becomes soluble and may be absorbed. The same remark applies to the products formed when

dialysed iron	is used to	neutralise arsenic,
tannic acid	,,	precipitate strychnine,
charcoal	,,	absorb morphine and other poisons,
French oil of turpentine	,,	oxidise phosphorus.

Solution of potassium permanganate decomposes morphine and many other alkaloids and other organic substances. The usual strength of the solution is 1 grain to the ounce, and even 4 or 5 ounces of this solution left in the stomach does no harm.

This principle of an antidote or neutraliser is so important that several attempts have been made to concoct one of universal application. The following formula[1] is probably the best, but is very incomplete :—

(1)	Saturated solution of sulphate of iron	100 parts.
	Calcined magnesia	88 ,,
(2)	Animal Charcoal	40 ,,
	Water	100 ,,

the two fluids to be mixed at the moment of using. Murrell states that it is ideal for arsenic, zinc salts, digitalis, and acids of the ordinary type, useful for mercury salts, morphine, and strychnine, but of no use for alkalies, phosphorus, tin salts, nor hydrocyanic acid.

3. **Apply Physiological Antidotes.** These agents act on the principle of antagonism, utilising, when possible, the power which different drugs possess of interfering with one another's action upon the cells of the body when brought into physical contact with those cells.

Such interference or antagonism may be due to the fact that the drugs combine chemically with one another, and that only the resultant

[1] Murrel: "*What to do in Cases of Poisoning.*"

combination which may be quite harmless acts upon the cell, or that the drugs influence the cells in different or contrary directions so as to alter or prevent the exhibition of that particular form of cellular activity which either individual drug would call forth, or that one drug may cause such alteration of the surface of a cell that another drug may be unable to be attached or absorbed.

It is possible to conceive of a complete antagonism between two drugs, but in practice we find that it is usually limited to the cells of one or two systems. The antagonism of *atropine* and *physostigmine* are seen in their opposite effects on the heart rate, state of the pupils, and glandular secretory activity. *Chloral*, *morphia*, and *aconite* depress the activity of the respiratory centre, while *strychnine* stimulates it. *Ergot* and *adrenaline* contract the blood vessels, while *nitrites* dilate them.

These are illustrations of antagonism, but in practice the principle is made use of only to a limited extent. Thus atropine is given hypodermically to counteract the action of morphine ; strychnine is used to stimulate the respiratory or cardio-vascular centre. The great obstacle to a more extensive use in poisoning cases is that of dosage. We do not know the amount of poison absorbed, and consequently are in the dark as to how much we should give of the counteracting poison. It is customary to give a full maximum therapeutical dose of the antagonist and watch its effects, administering another in half an hour or so if necessary. The completeness or the reverse of the antagonism is another difficulty, for the remedy in other directions may produce most undesirable results. For details of drug antagonism the reader should consult a modern handbook of pharmacology.

An interesting and outstanding example of a different type of physiological antidote is seen in the use within recent times of the compound known as B.A.L. (i.e., British Anti-Lewisite or 2 : 3 dimereaptopropanol) for poisoning by arsenic and other metals. This is discussed more fully in the section dealing with arsenic poisoning, but, expressed briefly, the principle is that a compound is introduced into the body which is similar to the tissue compound known to be attacked by the poison, in the hope that the poison will attach itself to the " antidote " and so spare the tissues.

4. **Keep the Patient alive by General Measures while his Eliminatory Organs are getting rid of the Poison.** Steps must be taken to combat the shock which is common in poisoning cases ; and any attempts made to counteract by *general* means the particular physiological (pathological) effect of the poison. Diffusible stimulants, artificial respiration, the inhalation of oxygen and carbon dioxide, the application of warmth to the trunk and extremities are illustrations of these general methods that may be adopted. Attempts should also be made by simple demulcents and anodyne applications to relieve the pain in mouth or stomach. The infusion of warm normal saline is useful in counteracting shock, and the addition of an alkali may be of value when the alkali reserve is diminished, as probably happens in many cases of poisoning. In many cases it is advisable to give glucose either by the mouth or by intravenous injection owing to the depletion of liver glycogen. The intravenous injection of solution of sodium thiosulphate in poisoning by arsenic and mercury is discussed under arsenic poisoning.

THE MEDICO-LEGAL ASPECT IN POISONING

This refers to the evidence which a medical witness may have to bring before the court to establish a charge of poisoning.

The questions which have to be decided are the cause of death and whether such death was caused by poison and not by disease.

To answer these the medical witness must possess full notes in writing concerning—

The symptoms during life.

The *post-mortem* appearances of the body and organs.

The chemical analysis (vomit, urine, stomach contents, organs, etc.) as to the presence of poisonous substances.

The Symptoms

It is impossible to discuss every conceivable symptom that might arise from the administration of poison, but there are certain points which specially demand the attention of the medical jurist. The following are the most important :—

(1) Their nature—especially vomiting, purging, convulsions, coma, etc.—the time of their occurrence, and the time relative to taking a meal or medicine. If present before, whether they were more violent after a meal or medicine or any particular food. The condition of the pupils, the pulse, the respiration, the skin and the mucous membranes.

(2) The order of occurrence of symptoms.

(3) Whether they were intermittent or continuous, becoming more aggravated up to death.

(4) Previous or contemporary known disease.

(5) When the physician was summoned, absolutely, and relatively to the occurrence of symptoms, and the reason for any delay.

(6) Voluntary explanations offered of the symptoms. These must be noted as nearly as possible in the *ipsissima verba* of the patient. Statements made by other persons present in the room who may be supposed to be concerned in the matter.

(7) Other persons affected, or others exposed to similar conditions without effect.

(8) Had the substance alleged to have caused the symptoms been taken previously without ill effect ?

(9) Any circumstances bearing on the question of suicide, accident, or homicide.

(10) Exact time of death, and the interval between the ingestion of the poison and death. If found dead, when was the patient last seen living or known to have been alive ?

(11) If the patient has vomited, the vomited matters, especially those *first* ejected, should be procured, their odour, colour, and acid of alkaline reaction noted as well as the quantity. If none are procurable, and the vomiting has taken place on the clothing, furniture, or floor of a room, then a portion of the clothing, sheet, or carpet, may be cut out and reserved for analysis. If the vomiting has occurred on a deal floor, a portion of the wood may be scraped or cut out, or if on a stone pavement, than a clean piece of material soaked in distilled water may be used to remove any traces of the substance. The vessel in which vomited matters have been contained will often furnish valuable evidence.

since heavy mineral poisons fall to the bottom, or adhere to the sides. Observe whether vomiting has taken place in the recumbent position or not. If the person has vomited in the erect or sitting posture, the front of the clothing will usually be found covered with the vomited matters.

(12) Endeavour to ascertain the probable nature of the food or medicine last taken and the exact *time* at which taken. Also ascertain the nature of all the articles of food used just before the symptoms appeared.

Ascertain if possible whether the patient noticed any change in the colour, taste or smell of the food or drink.

(13) Any suspected articles of food, as well as the vomited matters, should be sealed up as soon as possible in clean glass vessels, labelled, dated, and reserved for analysis. Observe all surrounding objects. Bottles, paper packets, weapons, or liquids lying about should be collected and preserved. Any obviously recent stains on bedclothes, furniture, etc., should be preserved if possible.

(14) *In preserving any specimens, whether vomit, viscera, or other articles, be especially careful to seal them up, label them, and keep them in such a manner and place that no one can tamper with them, and that they can be recognised and identified at any subsequent stage. A careful list must be made at the time.*

Post-mortem Examination

The patient may be dead on arrival or die shortly afterwards, and the following matters should be observed before making a *post-mortem* examination.

(1) The position and attitude of the body ; the condition of the clothing and any stains on or marks of destruction of the latter. Evidence as to whether a struggle had preceded death, or violent convulsions ; proofs of the nature of the poison—stains on clothing—or of alleged vomiting, diarrhœa, etc.

(2) The external appearance of the body, the colour of the *post-mortem* staining, whether the face is livid or pallid, whether the countenance is composed or distorted. These may be of importance in regard to the nature of the poison and its action. They may be corroborative of, or contradictory to, some other evidence. Note any signs of corrosion of the lips and about the mouth suggesting corrosive poisoning, and any smell about the mouth or nose suggesting volatile poisons.

(3) Any evidence of marks of violence, bruises, cuts, wounds of any nature ; they may suggest either a struggle or some form of death other than poison.

(4) The degree of warmth of the body (for choice taken in the rectum or vagina by means of a thermometer), also the presence or absence of *rigor mortis*. These points are of great value in determining the time of death (*vide* Vol. I). In noting the temperature, the circumstances in reference to rapidity of cooling must be observed, and similarly those regarding the onset of rigidity, which is sometimes influenced by poison.

(5) The time, both absolute and relative to the time of death, at which the autopsy is made—this has reference to signs of putrefaction —and any other points enabling the examiner to fix the time of death.

In relation to these *external* appearances, there are none which are specially indicative of poisoning upon which we can safely rely. The bodies of persons poisoned are not more rapidly decomposed, *cæteris paribus*, than those of others who have died a sudden and violent death from any cause whatever. Indeed, with some (antiseptic) poisons it is rather the reverse, for the poison inhibits the action of putrefactive bacteria (*vide* Vol. I, " Putrefaction ").

The Internal Appearances. There is no special order peculiar to poisoning cases in which the *post-mortem* should be done. The only thing is that *all* organs *must* be examined, and for convenience we may take the alimentary system first, for it is here, if anywhere, that special signs of poisoning are likely to occur.

It is particularly important, in making the autopsy, to prevent any soiling of the solid viscera with the contents of the stomach or intestine. It is wise therefore to remove these organs and place them in clean glass bottles before opening into the gastro-intestinal canal. It is as well also to secure samples of blood and urine at this stage and if samples of hair, nails, skin, bone or muscles are to be taken they should be removed before opening the stomach.

1. On opening the body be careful to note any **peculiar smell** that may be observable. Carbolic acid, hydrocyanic acid, phosphorus, chloroform, ether, and a few other poisons may be thus detected.

2. Examine the **tongue, mouth, and gullet** for any evidence of inflammation or erosion or staining. Also look for scars and stricture. A person had been known to die from a stricture of the gullet brought on by sulphuric acid *eleven months* after the poison was swallowed; and instances may occur of a still more protracted nature.

3. Examine the **larynx, trachea** and **bronchi** for evidence of volatile irritants.

4. Examine the **stomach,** noting the appearance of the mucous membrane and the contents.

(*a*) **Colour.** The colour and appearance may be normal; only corrosives and irritants are likely to alter the colour markedly. Only an extensive experience in the *post-mortem* room can enable a medical jurist to state what is the natural colour, but it is commonly pale, with only a few small vessels definitely visible on the mucous membrane, generally arranged stellately. If an irritant has been given, it is likely to be red, either in large patches not absolutely uniform all over or in smaller patches which can be shown to be due to foldings of the mucous membrane from spasm of the muscular layer. Genuine inflammation from an irritant is likely to show small hæmorrhagic foci, though these may be produced by violent vomiting due to causes other than administered poison. Caution is necessary in assigning irritant poisoning as the cause, for there is an important class of cases in which **redness** of the mucous membrane of the stomach is found after death **not dependent on the action of poison** or on any easily assignable cause. In many cases exposure of the mucous membrane to the atmosphere causes a reddening of the surface due to the absorption of oxygen by the blood. Such cases show none of the other inflammatory signs.

The redness of the lining membrane of the stomach, in cases of poisoning, is so speedily altered by putrefaction, when circumstances are

favourable to this process, as frequently to render it impossible for a witness to speak with any certainty upon its cause. Putrefactive infiltration from the blood contained in the adjacent viscera and muscles will give a reddish appearance to a stomach otherwise in a healthy condition. Great differences of opinion exist as to the length of time during which redness of the stomach produced by an irritant will be recognisable and easily distinguishable from putrefactive changes. No certain rule can be laid down on the subject; it must be left to the knowledge and discretion of the witness. In *R.* v. *Jennings* the well-marked appearances of inflammation produced by arsenic in the stomach and duodenum were seen in an exhumed body twenty-eight days after interment; and in other instances the reddened state of the mucous membrane, in the case of arsenical poisoning, was plainly perceptible on removing a layer of arsenic *nineteen months* and twelve months respectively after interment. If, however, there should be a reasonable doubt respecting the cause of redness, and no poison is detected, it would be unsafe to rely upon this appearance as evidence of poisoning.

Other colours than red may be present and due to definite causes. For instance, mercury usually causes a slate-coloured stain; arsenic may show white particles adherent, or possibly a yellow sulphide which has been found long after death; strong sulphuric acid and concentrated oxalic acid are likely to have blackened or charred the wall; nitric acid may turn it yellow (xanthoproteic reaction); carbolic or hydrochloric acid may have turned it buff coloured or white and shrivelled, though hydrochloric acid may blacken it. The congestion outside the stomach may be differentiated from hypostasis by stretching (*vide* Vol I). The colour may possibly be due to bile (yellowish red), when there will be no signs of inflammation, and the colour will be outside; or to fruit juice, when it is uniform and without signs of inflammation.

(*b*) **Ulcers.** If the corrosive has been sufficiently strong to produce ulceration there will be signs of intense inflammation, which should prevent such ulcers being mistaken for cancerous, chronic or acute simple ulcers, none of which in other respects look like those of corrosives, at any rate when the case is one of recent poisoning. When the poisoning is of older date it is impossible to differentiate the ulcer from a simple chronic ulcer by its appearance only, though the history and other circumstances of the ulceration may do so. Thus—

An examination was made in March of the stomach of a young adult who in the previous September had swallowed a corrosive by accident. His main symptoms had been gastric pain and vomiting ever since, and he died of exhaustion. The stomach was very thin and distended, chiefly upwards under the ribs. Over an area at the pyloric end of the stomach, roughly the size of the hand with the fingers together, was a stellate scar, with bands of thickening, very like the scar of an extensive burn on the skin. A second unhealed ulcer occupied the exact pyloric orifice and about an inch each of gastric and duodenal mucous membrane. There was in addition a scar with longitudinal lines and patches of thickening in the last three inches of the œsophagus.

It was possible from the superficial nature and large extent of the scar in the stomach to assert that it had come from a large superficial wound of the organ, and to say that this was probably attributable to a poison, because, if such an extensive ulcer had been attributable to disease, it would, according to all experience, have caused adhesion

between the stomach and the neighbouring viscera; and there was no trace of any such adhesion. The ulcer, still unhealed, actually in the grip of the pylorus, could not be distinguished by its appearance from one attributable to disease, but its position, extremely unusual in disease and common as the result of corrosives, was strongly suggestive. In the œsophagus, simple ulceration, with a consequent scar, is practically unknown.

In irritant poisoning the stomach is occasionally found ulcerated. In such cases the mucous membrane is removed in small distinct circular patches, under the edges of which the poison may be found. Ulceration of the stomach is a more common result of disease than of the action of poison. It is worthy of remark, as a means of distinction, that ulceration has never been known to take place from arsenic or any irritant poison until symptoms indicative of irritant poisoning have occurred, while the ulcers of disease are frequently latent until perforation or hæmorrhage leads to a fatal result. In ulceration from disease, the mucous membrane is commonly only reddened in the neighbourhood of the ulcer. In ulceration from poison, the redness is generally diffused over other parts of the stomach, as well as over the duodenum and small intestines. The history of a case previous to death will commonly enable us to determine to what cause the ulceration found may be due.

(*c*) Other Conditions of the Walls.

Actual perforation is occasionally witnessed when the strong mineral acids have been taken, especially sulphuric acid; it is much less common with the other acids. The stomach, in such cases, is blackened and extensively destroyed, the aperture is large, the edges are rough and irregular, and the coats are easily lacerated. The acid escapes into the abdomen, and may be readily detected there by chemical analysis. Of perforation caused by irritant poisons (arsenic) little is known, the recorded cases being but few. It is a rare appearance in cases of irritant poison. Nevertheless it is seen occasionally.

Perforation from poison must be distinguished from perforation from disease and also from digestive perforation. There should be no difficulty in the former case, for the ulcer of disease, when free from adhesions and thickening as in the acute cases which we are considering, never shows the signs of intense irritation which must be met with when poison has gone so far as to perforate, and in the majority of cases there are either old adhesions to other organs or thickening of the coats of the stomach from scarring. The aperture of perforation is different in the two cases: in poison it is ragged and irregular with torn edges; in disease it is more or less rounded and has a punched-out appearance.

Softening. The coats of the stomach are not infrequently found so soft as to yield and break down under very slight pressure; and this may be the result either of poisoning by alkaline irritants, or of the solvent action of the gastric juice after death. As this condition of the stomach, when caused by poison, is produced by those substances only which possess corrosive properties, it follows that in such cases traces of their action will be perceived in the mouth, throat and gullet. In softening from *post-mortem* digestion, the change will be confined to the stomach alone, and it is commonly found only at the cardiac or greater end of the organ.

Spontaneous or digestive *perforation* is not common, but *post-mortem* thinning or digestion of the inner coats is an almost constant phenomenon, and the question of actual perforation is merely one of degree (*vide* Vol. I). An inspection of the body, and the history of the case, will usually suffice to remove any doubt in determining whether the extensive destruction frequently found was caused by poison. Thus, in a cadaveric perforation, the aperture is usually situated in that part of the stomach which lies to the left of the cardia; it is very large, of an irregular form, and ragged and pulpy and softened at the edges, which have the appearance of undergoing the process of digestion (as, in fact, they are). The mucous membrane of the stomach is not found inflamed. There is occasionally slight redness, with dark brown or almost black lines (striæ) in and near the dissolving coats, which have an acid reaction. The chief point of distinction from the softening of poison is the complete absence of peritonitis in cadaveric perforation.

Hardening. This is not a frequent result of poisoning, but is occasionally found. The author has met with an instance in which the coats of the stomach were considerably hardened by sulphuric acid; and many observers have noticed that carbolic acid usually hardens the tissues with which it comes into contact.

(*d*) **The Contents of the Stomach.** The wall must be carefully examined for fragments of poison adherent to it. Bismuth, calomel, arsenic, fragments of leaves or fruit, cantharides, etc., etc., may possibly be found in this situation.

The contents must be carefully observed and written notes made with reference to

(*a*) Quantity.
(*b*) The nature of the food, etc.
(*c*) Colour, etc.
(*d*) The state of digestion.

} Of great importance for corroborating or contradicting evidence from other sources as to time and nature of last meal, presence of vomiting, etc., etc.

(*e*) The presence of matters not commonly considered as foods—*e.g.*, cantharides wings, berries, seeds, leaves or other parts of plants, bile, blood, and foreign bodies such as nails, pins, glass, etc.

(*f*) Odour. Many substances may be detected by their smell, for example opium, chloroform, carbolic acid, phosphorus, hydrocyanic acid or cyanides, benzene, camphor and alcoholic beverages such as beer, spirits or wine. If the contents have no smell in the cold, a small quantity may be warmed gently in a clean beaker, and the nose again applied. Nicotine, phosphorus (garlic smell), carbolic acid, etc., may thus become more apparent. It is well to get two or three independent persons to note the odour both in the cold and when the contents are warmed.

(*g*) The normal reaction of the gastric juice is acid. An alkaline reaction to litmus paper suggests poisoning by alkalies. A very strong acid reaction may suggest an acid poison, but if some poison is certainly present, and there are no gross naked-eye changes, an acid reaction makes it probable that the poison will require the prolonged search of the next section.

(*h*) Any other special characters not included in the above.

5. Examine the duodenum and its contents with the same care, and for similar purposes, as the stomach. A strongly acid reaction is of far more significance here than in the stomach contents. Poisons rarely cause duodenal ulcers, but inflammation is frequently met with, as in arsenical poisoning, for example. Inasmuch as the duodenum lies so near to the gall bladder, we must repeat the caution not to mistake a yellow or green discoloration produced by bile as a sign of inflammation or of poison.

6. Note the condition of the rest of the small intestine. It is very rare indeed for inflammation from irritants—*i.e.*, poisons—to reach far down the intestinal canal; extensive inflammation is more likely to occur from disease or from decomposed food. Ulceration of the small intestine is usually due to disease such as tuberculoses, typhoid fever, or possibly to stercoral ulceration.

7. The state of the large intestine and of its contents must next be observed. Inflammation of the cæcum is common in poisoning by corrosive sublimate. Arsenic seems to have a special effect upon the rectum, when no traces of it can be found between the duodenum and the colon. The discovery of hardened fæces in the colon and rectum tends to disprove a history of purging before death. Some care must be exercised in ascertaining that the purging was genuine, and not a mere false diarrhœa (mucus and blood with tenesmus), for in one case a colon and rectum was found full of hard scybala in a patient who was stated to have had diarrhœa before his death, which had nothing to do with poisoning. The point might be of very great importance in chronic or secret poisoning.

8. All the other viscera—kidneys, liver, lungs, heart, brain, spinal cord, uterus, ovaries, etc.—must be carefully examined for signs of disease of which the patient might have died, for negative evidence of this description is one of the first things to rouse a suspicion of poisoning in some cases that have not been carefully observed during life.

9. The bladder and (in females) the vagina should be particularly examined, for poison has been known to be introduced into the body by these channels.

The only details in the above manipulations that require special mention are—

Ligature the stomach at each end before removing it.

Ligature the duodenum at each end before removing it.

If either of these viscera is to be opened at once, place it unopened in a clean dish, so that it and its contents are not contaminated with unknown materials.

Similarly, before opening the bladder aspirate its contents into a clean bottle.

Take care that every viscus or material that requires to be saved is placed in a *clean* vessel, which must be securely sealed and also numbered or marked in such a way as to be identifiable.

It is advisable to take the whole of the viscera for analysis if this is possible. If it is not, the liver, kidneys and spleen must be removed, and such other specimens as may be necessary. It is advisable to take at least ten ounces of blood, especially if a volatile poison is suspected. **The solid viscera should not be contaminated with the contents of the alimentary canal.**

It is necessary to bear in mind on these occasions that the body is examined, not merely to show that the person has died from poison, but to prove that he has *not* died from any *natural cause*. Medical practitioners commonly give their attention exclusively to the first point: while lawyers, who defend accused parties, very properly direct a most searching examination to the last-mentioned point, *i.e.*, the healthy or unhealthy state of those organs which are essential to life, and with which the poison has probably not come in contact. The usual causes of *sudden death* have their seats in the brain, the heart and its great vessels, or in the lungs. Marks of effusion of blood, congestion, inflammation, suppuration, or a diseased condition of the valves of the heart, should be sought for and accurately noted, whatever may be the condition of the abdominal viscera. It is also to be recommended that an examination of the spinal marrow should be made. If the cause of death be obscure after the general examination of the body, there is good reason for inspecting the condition of this organ.

In cases of *chronic poisoning* there is sometimes great difficulty in assigning death exclusively to the original action of the poison, since the habits of life of the person, a tendency to disease, and other circumstances, may have occurred either to accelerate or produce a fatal result. To connect a stricture of the gullet (proving fatal) with the effects of poisoning by a mineral acid it would be necessary to show that there were no indications of the disease before the acid was administered; that the symptoms appeared soon after the first effects of the poison went off; that these symptoms continued to become aggravated until the time of death; and, lastly, that there was no other cause to which death could with any probability be referred. These remarks apply equally to the secondary fatal effects of any poisons, such, for instance, as the salivation occasionally induced by corrosive sublimate, and the exhaustion and depression which are caused by tartar emetic, when the acute symptoms of poisoning by these substances have passed away.

It is important, as a last general remark on the autopsy, to bear in mind that, except for corrosives and irritants, **it is extremely easy and common for poison to destroy life without leaving any naked-eye changes**, and proofs of poisoning must be derived from other sources or from the chemical examination.

THE CHEMICAL ANALYSIS AND THE DUTIES OF THE EXPERT ANALYST

Before undertaking to appear in court the expert analyst must be prepared to answer the following questions, for the strength of a chain is that of its weakest link, and many charges of poisoning have broken down through failure to notice an apparently irrelevant detail.

1. **When, where, and from whom did you receive the Articles which you have analysed?** These are but elementary and obvious questions in regard to the identity of substances received; they only emphasise the importance of immediately making written notes upon such serious matters.

2. **In what State were they received?** *Were they secured in any way?—if so, how?—or were they exposed, wrapped in paper, rag, etc., in an open*

box or tin, etc.? Did you receive more than one object? If so, how many? Was each separately labelled, or in a separate receptacle? What quantity was there of each by weight or measure?

It is necessary to observe that all legal authorities rigorously insist upon proof being adduced of the *identity* of all substances taken from the body of a deceased person when poisoning is suspected. If during the examination the stomach and viscera are removed from the body, they should never be placed on any surface, or in any vessel, before first ascertaining that the surface or vessel is perfectly *clean*. If this point be not attended to a doubt may be created in the minds of the jury whether the poisonous substance might not have been accidentally present in the vessel used. This may be regarded as a very remote possibility; but, nevertheless, it is upon technical objections of this kind that acquittals take place in spite of the strongest presumptions of guilt. This is a question for which every medical witness must be prepared, whether he is giving his evidence at a coroner's inquest or in a court of law. Many might feel disposed to regard matters of this kind as involving unnecessary nicety and care, but if they are neglected it is possible that a case may be at once stopped, so that the care subsequently bestowed upon a chemical analysis will be labour thrown away.

Evidence of the presence of poison in the contents of a stomach was once rejected at a trial for murder because they had been hastily thrown into a jar borrowed from a neighbouring grocer's shop; and it could not be satisfactorily proved that the jar was clean and entirely free from traces of poison (in which the grocer dealt) when used for this purpose.

When the life of a human being is at stake, as in a charge of murder by poisoning, the slightest doubt is always interpreted as far as possible in favour of the accused.

Not only must clean vessels be used for receiving any substance destined for subsequent chemical analysis, but care must be taken that the *identity* of the substance is preserved, or the results of analysis may be inadmissible as evidence. The suspected substance, when once placed in the hands of a medical man or analyst, should never be allowed to leave his custody. It should be kept sealed under his private seal, and locked up while in his possession, in a room to which no other person can have access. If he has once allowed the article to pass out of his hands into the hands of several other persons, it may become necessary for those persons to be called to give evidence as to what happened to the article while it was in their possession. The exposure of a suspected substance on a table, or in a closet or room to which many have access, may be fatal to its identification. (*Melius est absolvere reum quam damnare innocentem.*)

The importance of this is emphasised by the following notes.

Mr. Justice Grantham deprecated the practice of passing articles for chemical examination through so many hands. Mr. Justice Hawkins made a similar complaint in the Lamson case. In the Flannigan and Higgins case a most important piece of evidence was nearly excluded because a police official was lax in preserving the identity of an important article. The moral is, let the medical witness himself secure and lock up everything which ought to be sent to the analyst. This will save time, trouble, expense and prevent a possible miscarriage of justice. (*Lancet*, 1896, I, p. 586, *in re* the Huddersfield case of poisoning by white precipitate.)

In one case, an analysis of certain matters vomited by a person poisoned with arsenic was not admitted as evidence against the prisoner because the medical man had left them in the custody of two women; and these women had allowed the vessel containing the suspected liquid (which was proved to contain arsenic) to be exposed in a room open to the access of many persons. In another case, the analysis of some suspected liquids was not allowed in evidence because the practitioner, who lived in the country, and was unwilling to take the responsibility of analysing them, had sent them up to town by a carrier, to be examined by a chemist.

In *R.* v. *Hearne*, bottles containing viscera were left uncovered in a graveyard, the soil of which was heavily contaminated with arsenic.

When any article is reserved for analysis, care should be taken at once to attach to it, or to the sealed vessel containing it, a label, upon which are plainly written in ink the name of the deceased, the nature of the material, and the date, including the day of the week and month. This is especially necessary when there are two or more articles for analysis. The greatest inconvenience has resulted from the neglect of this simple precaution.

Preserving Articles for Analysis. In removing viscera or liquids from the body, and preserving them for analysis, it is necessary to observe certain precautions. A clean vessel with a wide mouth should be selected; it should be only sufficiently large to hold the organ or liquid (the less air remaining in it the better); it should be secured by a closely fitting glass stopper. If a refrigerator is available all organic substances should be placed in it as soon as possible after removal from the body. Decomposition to some extent cannot usually be prevented, for antiseptic chemical compounds must not be added lest they should confuse the analysis. It is true that decomposition may produce bodies not in the original stomach, but it is within a skilled analyst's power to make allowance for these without confusion. If the mouth of the vessel be too wide for a stopper, it must be properly closed with some airtight material. Paper only should not be used: the appearances after death of the viscera of an infant, suspected to have died from poison, have been entirely destroyed by drying from the evaporation which took place through the layers of paper with which the vessel in which they were contained was covered. The practitioner should bear in mind that all these matters are likely to come out in evidence; and whatever is worth doing at all is worth doing well.

The articles used for the preservation of viscera should in all cases be scrupulously examined. Some kinds of calico are dressed with arsenic and starch paste, and many kinds of wrapping paper as well as wall-papers are strongly impregnated with this poison. The following account shows that this is not an unnecessary caution.

[A pathologist was engaged in examining the body of a child, in order to determine the cause of death. The organs were healthy, and as no sufficient cause presented itself, he removed the stomach with a view to making an analysis of its contents. He was suddenly called away; and, to preserve the stomach, he wrapped it in a piece of paper (used for papering rooms), placed it on the uncoloured side, and locked it in a cupboard until the following day. When he analysed the contents, he found a trace of morphine with a large quantity of arsenic. As the symptoms from which the child had suffered were not those of poisoning with arsenic, and there were no appearances of the action of this substance on the body, he came to the conclusion that there must be some extraneous cause to account for its presence. He examined a portion of the wall paper in which the stomach had been wrapped

and then found that that part of it which was coloured yellow was tinted with arsenious sulphide or orpiment. Since commercial orpiment contains white arsenic, it was evident that the stomach and its contents had imbibed a portion of the poison during the night.

This satisfactorily accounted for the presence of arsenic in circumstances which might have given rise to a false charge of murder. Many wall-papers, having a tinge of green or yellow, contain arsenic, and this arsenic spreads by imbibition to other parts of the paper not so tinted. It is advisable in all cases to avoid the use of any coloured wrapper.

The results of an analysis, in the shape of sublimates or precipitates, should be preserved as evidence, distinctly labelled in small glass tubes, hermetically sealed. They can then, if asked for, be produced for examination at the inquest or trial.

3. **When did you analyse it, and where ?** These questions obviously tend to criticise the circumstances in which the analyst kept any material entrusted to his care. They raise the possibility that delay might have afforded opportunity for fraud, mischief, or decomposition, or that the equipment available was not adequate for a proper analysis.

4. **Did you analyse it alone, or were you assisted ?** *If so, by whom and to what extent ?* These questions obviously are directed at the competency of the analyst and his assistants. They also may raise a suspicion of the *bonâ fides* of the assistants and the opportunities any of them may have had for interference by carelessness or accident.

5. **What Tests did you employ ?** In a written report these need not be minutely described : a general outline of the analysis will suffice ; but in court defending counsel may cross-examine an analyst very closely on the tests he employed. As corroborative tests applied to comparatively pure substances, every analyst will have several to which he may attach special importance or with which he may be peculiarly familiar, but there are certain general or preliminary procedures which are practically universally adopted when gross masses of animal remains [viscera and (or) their contents] are presented to an analyst that he may determine the presence in them of poison.

GENERAL METHODS FOR THE SEPARATION OF POISONS

A search for a completely unknown poisonous substance is an extremely laborious process, and, if it were carried out in every case, would involve an enormous waste of time and material. Wherever possible the analyst should either be directed to search for specified substances or groups of substances, or should be given all the available information, so that he can, by proper use of it, direct his efforts into the most profitable channels. Notwithstanding such assistance, however, it is the duty of the analyst so to use his material and so to order his work, that if one line of enquiry should yield negative results, he still has the means of pursuing others.

For the purposes of chemical analysis, poisons may be classified according to the methods used for their separation from the tissues, although the classes are not mutually exclusive. Such a classification

does not coincide either with the pharmacological classification (p. 230) or the compromise classification given on p. 289. It is convenient to distinguish four main groups as follows :—

1. Substances separable by distillation or steam distillation (*i.e.*, volatile poisons) : a group which includes alcohols, phenols, aldehydes, some acids, hydrocarbons and their halogen derivatives, phosphorus, essential oils, etc.

2. Organic poisons soluble in alcohol : a group divisible into two sub-groups, one including those non-basic substances such as glycosides, barbiturates, etc., which are generally extractable from aqueous acid solution by chloroform or other immiscible organic solvents, and the other including bases (alkaloids) which are similarly extractable from alkaline aqueous solutions. [These sub-groups overlap ; some substances can be extracted from either acid or alkaline solution though not, as a rule, with equal ease].

3. Toxic metals : a group within which may be distinguished a sub-group often termed " heavy metals," and consisting of those which are deposited on copper foil when their salts are boiled with hydrochloric acid with copper foil suspended in the solution.

4. Substances requiring special methods of extraction, identifiable only by derivatives or by their effects, and substances not identifiable by any existing methods. This group includes various gaseous poisons like carbon monoxide, toxic proteins like ricin, vegetable products of unknown constitution, and so on.

Ideally, a general method of separation would allow a complete search to be made, one group being separated or eliminated in such a way as to leave it possible to examine the residual material for the remaining groups, and so on. Fortunately, given proper co-operation between the analyst and the medical-jurist, or other person who has furnished the samples for analysis and the relative information, such a complete search is rarely necessary. Even when it is, if sufficient material is available, it is usually possible and is certainly more convenient to examine different parts of the material for different groups. In the extreme case, however, it would be possible first to examine for volatile poisons, than to use the non-volatile residue for the extraction of the alcohol-soluble organic poisons (with the reservation that a few unstable poisons might be destroyed by the previous heating) and finally in the alcohol-insoluble matter, to search for toxic metals. Substances in group 4 capable of being identified would, even then, need to be sought for in separate samples of the material.

Volatile Poisons

Since the material to be examined—viscera, excreta, etc., contains a good deal of water, there is no convenient method of separating the strictly volatile substances from those which are only volatile in steam, and it is, therefore, convenient to use a steam-distillation apparatus at once. All apparatus, as indeed in all toxicological analysis, must of course, be scrupulously clean, and the final cleansing should be done by the analyst himself as the responsible person ; new apparatus should be cleaned just as carefully as that which has been used on previous

occasions. The flask should be of the short-necked or " bolt-head " type, the condenser of the straight, Liebig type, the tube from the condenser should dip well into the receiver which should be immersed in ice, and should have its neck loosely plugged with cotton-wool or glass-wool. It is convenient to make the distillation in two stages—first with the material (which in the case of solid viscera, etc., will have been chopped into small pieces or minced) as received, and later after acidification with dilute sulphuric or tartaric acid ; in this way it may be possible to distinguish whether a toxic volatile acid has been present as such or as a non-volatile salt.

Tests for the various substances which may be present will be found in later sections of this book, but it should be borne in mind that, as has already been mentioned, the classification given at the beginning of this section does not provide a clear-cut division into mutually exclusive groups. Thus a few alkaloids (*e.g.*, nicotine), are steam volatile, and so are some metallic compounds (*e.g.*, some derivatives of arsenic, mercury, zinc, etc.).

Organic Poisons Soluble in Alcohol

The method preferred by most toxicological analysts is that originally devised by Stas, modified by Otto, and many later chemists to obtain more complete extraction and better fractionation usually known as the *Stas—Otto process*. Recently, attempts have been made to elaborate simpler and surer methods, but as yet insufficient work has been done to demonstrate the general validity of these, although some success has been achieved in special cases ; they will be noticed later, but the Stas—Otto method, in view of its general use and, indeed, its general utility, must remain the safest general procedure, and must therefore be described first.

The Stas—Otto process depends upon the facts : (1) that whereas, almost without exception, the organic poisons are reasonably soluble both in very dilute aqueous acid and in warm ethyl alcohol, the protein and most of the fat in animal tissues is insoluble in alcohol ; (2) the organic poisons can be extracted from aqueous solution or suspension by various solvents which are immiscible with water, the neutral or acidic poisons being extracted from acid aqueous solution, the basic poisons from alkaline.

The First Extraction with Alcohol. The material to be examined may consist of :—

(*a*) Liquid or semi-liquid discharges, such as urine, stomach-wash, vomited matter or fæces, or (*b*) viscera.

Liquids should first be evaporated almost to dryness at a low temperature, and the resulting residues treated in the same way as solid materials.

The viscera to be examined are finely divided by cutting or mincing and mixed with about twice their weight of alcohol in a large Erlenmeyer flask. It is sometimes advised to use alcohol diluted with about half its volume of water, as pure alcohol, or rectified spirit, tends to form a lumpy coagulum, from which it is difficult to extract the alkaloids completely. With care in the mixing, however (the alcohol should be added slowly with constant stirring or shaking) this danger can be avoided, and the use of rectified spirit at this stage undoubtedly shortens the process.

A small quantity of a weak organic acid, tartaric, citric, or acetic, is added, so that the liquid, after shaking, reddens litmus paper. Mineral acids should not be used, as they tend to bring into solution large amounts of material which render the subsequent processes more difficult and laborious, and may, besides, destroy some of the less stable substances. The neck of the flask is loosely plugged with pure cotton wool or (better) fitted with a long tube to act as a reflux condenser, a thermometer is placed with its bulb in the liquid, and the mass is warmed on the water bath. The less stable alkaloids, such as atropine and aconitine, are liable to be hydrolysed either by a too strongly acid solution or by too high a temperature, and if there is reason to suspect the presence of these alkaloids the temperature should not be allowed to rise above 35° C.

Otherwise, it is recommended to raise the temperature to 50° C. for about an hour, then allow the mixture to cool for a few hours, and filter off the liquid through a double piece of muslin. The filtration should be carried out with the aid of gentle suction, using a Buchner funnel and flask, so that the residue can be thoroughly pressed. The pressed residue is again extracted with warm alcohol, the mixture being thoroughly shaken or stirred to loosen the caked material and, by giving an even suspension, to ensure proper extraction. It is often a help to re-mince the material before extraction. The mixture is strained and pressed when cold. This process is repeated a third time, the collected filtrates are allowed to stand a few hours and are then filtered with *gentle* suction. Strong suction should on no account be used as it causes the pores of the filter paper to become choked and so slow down or even stop the filtration. Reasonably rapid filtration can be obtained without suction by the use of a coarse, fluted paper. The clear filtrate is then evaporated to dryness.

Evaporation. At this stage also the danger of hydrolysis must be borne in mind, and the temperature must be kept low. The most rapid means of removing the alcohol and water is by distillation under reduced pressure. With an efficient water-pump this can be accomplished without the temperature rising above 40–45° C. On no account, unless it is certain that only the more stable alkaloids are present, should it be allowed above 60° C. Distillation *in vacuo* may, with extracts from very fatty material, be difficult because of the tendency of the liquid to froth. This tendency can be overcome by addition of a few drops of octyl alcohol, provided this substance is itself removed at the end of the distillation—if necessary by passing a current of air through the flask. A simpler but slower plan is to place the alcoholic solution in a shallow porcelain dish and allow a current of heated air to play on the surface. The evaporation is continued until the residue forms a thin syrup or paste.

Precipitation of Proteins and Fats. It is required to dissolve the salts of the alkaloids or other poisons in alcohol and to cause the fatty and albuminoid impurities to separate in a granular form. Great care is necessary at this stage, otherwise very undesirable sticky masses may be formed. A small quantity of warm absolute alcohol should be poured on to the surface of the residue, and the vessel gently rotated until the alcohol is turbid. This liquid is poured off into a dry wide-mouthed jar, and the operation is repeated with further quantities of absolute

alcohol until the residual mass begins to break up. Then, and not until then, a stirring rod may be used to loosen the cake and break it up. If the residue is large a considerable quantity of absolute alcohol may be necessary to effect the separation of the impurities. The jar containing the material is closed with a stopper and allowed to stand, with occasional shaking, for twenty-four hours, after which it is filtered with suction and the residue washed on the filter with absolute alcohol.

This filtrate is evaporated to dryness as before, and if the residue should be unduly large or obviously contain large amounts of fat, the process of taking up in absolute alcohol ought to be repeated. It is in fact, advisable to continue the process of extraction with absolute alcohol until an extract has been obtained whose residue after evaporation yields no appreciable precipitate on addition of an equal volume of alcohol. Care should be taken, however, by evaporating as completely as possible at each stage, to minimise the number of extractions necessary to produce this result. Every successive extraction causes some loss of material, and the quantities of poison present are usually so small that loss can ill be afforded. In obstinate cases where large residues remain on evaporation, it may be advisable to add a further quantity of organic acid to the alcohol to assist the precipitation before putting the jar and its contents to stand. But this should be avoided if the presence of the more unstable alkaloids is suspected.

Alternative Method of removing Fatty Impurities. The residue obtained by evaporating the first alcoholic extract is taken up in acidified water and allowed to stand in a tall vessel overnight in an ice chest. The aqueous liquid is then separated, evaporated to dryness, and the residue is taken up in absolute alcohol as previously described.

When the quantity of fatty and albuminoid matters has been thus reduced as far as possible, the residue is stirred with a little warm water, a few drops of dilute sulphuric acid are added, and the mixture is transferred to a filter. After filtration the residue is washed with water and the washings are added to the filtrate.

This filtrate now contains salts of any alkaloids present, together with traces of impurities not yet removed.

The Shaking-out Process. The salts of the more strongly basic substances remain in aqueous solution in presence of acid on shaking the latter with an immiscible solvent, such as chloroform or ether, but some of the less strongly basic substances together with neutral and acidic matter will be extracted by the added solvent. Various organic solvents have been recommended for dissolving out the residual impurities together with the non-basic or weakly basic matter, but chloroform is in general to be preferred to any of them. Although many workers prefer chloroform, there are advantages in using ether for this first extraction from acid aqueous solution, since it removes less of the weakly basic alkaloids. On the other hand, ether is not a good solvent for many glycosides, and if only one solvent is to be used, chloroform should probably be the choice. Much will depend on the particular substance or group of substances which is being sought, but if the analysis is a general one (*i.e.*, if there is no guidance, and a complete search for all possible organic poisons is being attempted) there is much to be said for a fractional extraction by a series of solvents. If this method is to

be used, the acid aqueous solution is extracted first with petroleum ether (which removes essential oils), ether (which takes out hypnotics and soporifics—barbiturates, other ureides, sulphonal, etc.), and chloroform (which dissolves glycuronides and weakly basic alkaloids such as ergot as well as one or two alkaloid salts such as those of heroin); with each solvent three or four extractions should be made. The method of extraction is as follows:

The acid aqueous solution is poured into a separating funnel, and about one-third of its volume of the immiscible solvent is added. There is a danger of forming a stable emulsion on shaking the two liquids together and, therefore, the shaking with the first quantity of solvent should be cautious. It is true that by using organic acid in the preliminary extraction and by thorough removal of fat and protein in the intermediate stages, this danger is small. Nevertheless it exists, and care must be exercised accordingly. If an emulsion should be formed in spite of careful shaking it may be broken by (*a*) rotating the funnel briskly, (*b*) stirring with a glass rod, (*c*) adding more solvent, or (*d*) centrifuging. When the two liquids are completely separated, the lower layer is gently run off, and a second quantity of solvent is added. The process of shaking and separating is repeated with this and a third quantity of solvent.

If, as sometimes happens, semi-solid matter separates in the aqueous layer during this process, it is advisable to remove this by filtration before proceeding to the next stage. In any case, the ether or chloroform extracts should be shaken with a little water, in a separating funnel, and the washings returned to the main aqueous solution.

The mixed chloroform extracts from the acid liquid may contain various substances, such as glucosides, weakly basic alkaloids, picrotoxin, sulphonal, veronal, etc. (partially separated, however, if fractional extraction has been done), whereas alkaloids in general remain in the aqueous portion.

A weak alkali, such as sodium bicarbonate or ammonia, is added to the aqueous liquid until the latter is distinctly alkaline to litmus, and the free alkaloid may now be extracted by shaking with a suitable solvent. Chloroform is the most generally useful solvent, but either hot amyl alcohol or a mixture of ether and ethyl acetate (in equal parts) is most suitable for morphine, which is only very slightly soluble in chloroform.

Three extractions at least with the immiscible solvent should be made, and the resulting extracts, after being washed by shaking with water, put in a warm place to evaporate. The residue, if any, should contain the greater part of alkaloid present in the original material with certain exceptions. These are the feebly basic alkaloids, such as those present in ergot, which are said to be extracted more or less completely by chloroform from the aqueous acid solution. The residue should be dissolved in water, acidified with sulphuric acid, and drops of the solution should be tested with three or four of the general alkaloidal precipitating reagents.

If these indicate the presence of an alkaloid, the residue should be purified before proceeding to further tests.

Purification of the Extracted Alkaloids. The acid solutions of the alkaloids remaining after evaporation of the chloroform is filtered and the residue washed with water. The filtrate is then subjected to the shaking-out processes with immiscible solvents, as before. The acid solution is extracted three times with chloroform, and after making alkaline the liquid is extracted three times with the chosen solvent. This final alkaline extract is then evaporated at a low temperature.

Since it is useless to perform the specific tests for the recognition of alkaloids unless the residue is in a state closely approaching purity, it may be necessary to repeat this process of purification. In any event the residue should be again tested with the general precipitating reagents. It frequently happens that the precipitates obtained by the action of these reagents on the crude extract are due to non-alkaloidal impurities (*e.g.*, protein), and in such cases the general tests on the purified extract will give negative results.

In the special case of strychnine a more drastic and efficient method of purification is available, since this alkaloid is a remarkably stable compound. The crude residue may be covered with strong sulphuric acid and heated on a boiling-water bath for two hours or more, so as to char or otherwise decompose the impurities present. The charred mass is extracted with water and filtered. After making the filtrate alkaline, the alkaloid may be re-extracted with chloroform.

Purification of the Non-Basic Substances. It may be necessary to examine the residue obtained by the evaporation of the chloroform extract from the acid solution, *i.e.*, the first extract made with an immiscible solvent.

The purification of this residue presents more difficulty than that previously described. If chloroform extraction alone has been employed, the combined extracts are evaporated to dryness, and, the residue (which may be oily), is dissolved in hot water slightly acidified with dilute sulphuric acid. This solution or suspension is thoroughly extracted with petroleum ether, the extract being evaporated and the residue (if any) tested for the presence of substances with vesicant action. The aqueous residue from the petroleum ether extraction is next extracted with ether; the extract is evaporated and the residue, if any, is tested for the presence of hypnotics and soporifics (barbiturates and other ureides, sulphonal, etc.). The aqueous residue is submitted to a third extraction, this time with chloroform or some special solvent such as amyl alcohol and the residue from the evaporation of this extract is tested for glycosides.

If the material has already been fractionated, the various extracts are evaporated to dryness, tested for the possible constituents and, if necessary, further purified by being redissolved or resuspended in very dilute sulphuric acid and re-extracted with the appropriate solvent.

Certain specific non-alkaloidal poisons lend themselves to special modes of purification, as in the lead acetate (Palm's) method for the purification of residues containing picrotoxin (*q.v.*).

General Notes. Throughout the process great care must be given to the washing of residues after filtration and adding the washings to the filtrates. The chloroform with which the acid aqueous liquor has been shaken should also be washed by shaking with water in a separating

funnel and the washings added to the main bulk of the aqueous liquid before adding alkali.

Considerable doubt has been expressed as to the possibility of making such a process of extraction yield even approximately quantitative results, since the separation depends on the supposed insolubility of the salts of alkaloids in immiscible solvents, and the insolubility is never absolute; or, to speak more strictly, a certain degree of salt hydrolysis may always be expected in aqueous solution, and the base must dissolve to a greater or less extent in the immiscible solvent, even in the presence of acids. The possibility of the adsorption of the alkaloids by the residues of the tissues and the precipitated proteins remaining on the filter should also be borne in mind.

The following are some of the substances which may be sought for in the chloroform extracts from the acid and the alkaline aqueous liquors respectively :—

(*a*) From the acid liquor : Piperin, picric acid, salicylic acid, benzoic acid, camphor, ethereal oils, caffeine, geissospermin, cantharidine, elaterin, colocynthin, digitalin, colchicin, papaverin, narcein, hydrastin, acetanilide, picrotoxin, gelsemic acid, the alkaloids of ergot.

(*b*) From the alkaline solution : Practically all the remaining alkaloids. Morphine requires a special solvent, *viz.*, hot amyl alcohol or a mixture of ethyl acetate and ether.

The following are **the common alkaloidal reagents** :—

Picric Acid Solution. A cold saturated aqueous solution of the acid.

Phosphomolybdic Acid. Sodium phosphomolybdate is dissolved in ten times its weight of a mixture of 1 volume of strong nitric acid (specific gravity 1·42) with 9 volumes of water.

Mayer's Reagent, or potassium mercuric iodide. It is prepared by dissolving 1·35 grammes of crystallised mercuric chloride and 5 grammes of potassium iodide separately in water, mixing the solutions so obtained and diluting to 100 c.c.

Iodo-potassium Iodide. Prepared by dissolving 10 grammes of potassium iodide in about 50 c.c. of distilled water, adding 5 grammes of iodine and, when this is dissolved, diluting to 100 c.c. with distilled water.

Tannic Acid. A 5 per cent. aqueous solution of tannin.

For the precise identification of the substance or substances extracted, it is necessary to apply the appropriate tests either for the particular compound sought or according to some systematic scheme, such as that given by Bamford.[1]

Other Processes

Two processes for the extraction of non-volatile organic poisons, though not yet sufficiently used to be accepted as of universal applicability may be described as showing the trend of research and as being reliable at least for the specific substances mentioned by their authors.

Stewart, Chatterji and Smith[2] obtain a clear filtrate, free from fat and protein by carefully grinding the minced material with an equal weight of 10 per cent. trichloracetic acid solution, allowing the thin paste so

[1] "*Poisons: Their Isolation and Identification.*" Churchill, 1940.
[2] *B.M.J.*, 1937, October 23rd., 793.

obtained to stand for half an hour or longer, and then filtering with suction. The residue, pressed as dry as possible, is re-extracted with trichloracetic acid and finally with water. The combined extracts contain all the alkaloid present (strychnine, morphine, quinine, nicotine, aconitine have been tested) and in tests, barbiturates and certain glycosides were also found to be extracted. From the filtrate, the poison is absorbed on kaolin which has previously been well washed with alcohol, chloroform and ether, and then re-dried, 10 g. of the kaolin being used for the extract (usually about 800 c.c.) from 400 g. of material, and half an hour or longer being allowed for the absorption. From the kaolin the poison is extracted by a suitable organic solvent, without neutralisation for the non-basic substances and after treatment with alkali for the bases. The wet kaolin may be shaken with hot chloroform and the chloroform filtered off and separated from the small amount of water which comes with it ; this process is then repeated after the kaolin has been suspended in dilute sodium carbonate solution. Alternatively, the kaolin may be ground with anhydrous sulphate and the dry powder so obtained extracted in a Soxhlet apparatus (for non-basic poisons) or similarly treated after being made alkaline with sodium carbonate (for alkaloids).

Daubney and Nickolls[1] dislike trichloracetic acid which, unless carefully used, gives a protein precipitate difficult to filter, and considered absorbents to be insufficiently effective. They freeze the material, mince it while still frozen, mix to a thin paste with dilute acetic acid, and saturate with ammonium sulphate. The mixture is warmed to 65° C. until the protein coagulates, when it is filtered off and washed with warm water. The residue is re-extracted by maceration with 1 per cent. acetic acid, and this process is repeated until the combined filtrates total 1,000 c.c. from 200 g. of material or 1,500 c.c. from 400 g. The combined filtrates are then extracted five times with chloroform, 100 c.c. for each extraction. The alkaloid is re-extracted from the chloroform by 3 N. sulphuric acid (three extractions) and water (once) and these mixed extracts are in their turn made alkaline with ammonia and extracted five times with chloroform. This extract is evaporated to dryness and submitted to further purification for weighing and testing.

Separation of inorganic poisons

In effect, the inorganic poisons to be considered in this section consist of derivatives of the toxic metals or metalloids—arsenic, antimony, bismuth, copper, silver, mercury, lead, barium, chromium (chromates and bichromates) manganese (permanganates), etc. The non-metallic poisons like phosphorus, chlorine, the so-called mineral acids, boric acid, etc., are identifiable by special means and, since they produce such definite effects, do not need to be included in a search for more or less obscure poisons.

An essential part of any toxicological analysis unguided by definite instructions is the Reinsch test which affords a delicate means of detecting those metals which, from acid solutions of their salts, can be deposited on metallic copper—a group which includes arsenic, antimony, bismuth and mercury. The technique of this test is fully described in the section on arsenic (p. 430) where are also given the means of differentiating

[1] *Analyst*, 1937, 62, 851. 1938, 63, 560.

the various types of deposit and so identifying the particular metal responsible. A positive result in the Reinsch test is an indication for the application of other specific confirmatory tests; a negative result, however, does not mean that the search for certain minerals can be abandoned, but only that still more sensitive tests must be employed for, delicate as it is in relation to some other tests, the Reinsch test is crude compared with, *e.g.*, the Marsh or the Gutzeit test for arsenic.

In any case, it will become necessary, sooner or later, to use some process which will destroy the organic matter present, and leave the inorganic constituents of the material intact and in a form capable of yielding a solution to which the various tests can be applied. Two processes are available—the wet and the dry. The latter consists simply in heating the material in a platinum or silica crucible until the whole of the organic matter has been oxidised and the residual ash can be dissolved in nitric acid. Since some metallic salts are volatile at, or even below, the temperature necessary for rapid combustion of the organic matter, this process is dangerous if the identity of the possible poison is unknown; and even when it is employable, the heating should be done at as low a temperature as possible.

The **wet process** consists essentially in heating the material with a solution containing a powerful acid-oxidising agent, thereby oxidising the organic matter without the necessity of using very high temperatures, and leaving the inorganic matter in the form of a solution of salts. Various mixtures have been employed for this purpose—hydrochloric acid with potassium chlorate or perchlorate, a mixture of concentrated sulphuric and nitric acids, dilute acid and "100 vol." hydrogen peroxide, etc. Of the agents named, the second offers many advantages for general use. Whichever is used, however, the method is much the same, the material is mixed with the less active agent (hydrochloric or sulphuric acid) and to the heated mixture the more active agent is added a little at a time, the heating being continued gently after each addition until the reaction is finished, and being so controlled as to avoid too violent an action. The process is continued until the solution which, intermediately, will be brown or black, becomes colourless or pale and does not darken when heated with a little more of the oxidising agent. Finally, the solution is boiled to remove excess of the oxidising agent or its decomposition products. Metals like arsenic which form two series of salts will, by this process, be converted to a salt of the higher series, and since, in the case of arsenic and antimony, it is only salts of the lower series which respond to the usual tests, it is necessary, before applying these tests, to treat the solution with a suitable reducing agent (*e.g.*, sodium or potassium metabisulphite) which will give a soluble salt of the lower series.

The **Dry Process,** as stated, consists in burning the material in a platinum or silica dish at as low a temperature as will serve the purpose. The resulting ash is extracted with strong nitric acid and filtered. The metal is thus obtained in solution as a nitrate, and can be tested for by the ordinary methods (*vide* caution under "Tests for Lead").

Having thus considered in some detail the various methods or types of test which the analyst may employ, it is necessary to continue a consideration of those further questions which he may be asked in court even after he has established that a poison or "noxious agent" was detected by his analysis.

6. **If Poison was found, was it pure or mixed ?** This question may be put to confuse an expert witness or to see what his chemical knowledge may be in regard to the chemical relationships of any poison. It may have reference also to the presence of soot or other colouring matter showing the commercial source of the poison.

7. **What was the Strength or Percentage of Poison found and also the Absolute Quantity ?** These are obviously important questions with reference to the dose required of the poison either to kill, to be a " noxious thing," or to annoy. It may also have an effect on motive.

Again, the importance of the answer would be very much affected by the locality where it was found, *e.g.*, in a vomit, in an external article, or in an organ such as liver or brain.

8. **What is the Dose necessary to kill ?** *Ab initio* this is a matter of speculation and experiment on animals, but later becomes a question of record from previous cases. We have already (pp. 230 *et seq.*) drawn attention to the difficulties of dosage as a matter of exact scientific proof. In court cases, the minimum dose known to have been swallowed by a person who died as the result of the dose, is accepted as the minimum fatal dose.

9. **In what Organs or in what Material did you find it ?** This has a strong bearing on the previous two questions. If the poison is only found in the contents of the stomach, and none in the solid viscera, and is not an irritant, grave doubts may be thrown on the statement that it was the actual cause of death. Poison found in a viscus (other than the stomach or intestine) is proof of absorption ; hence the importance of keeping the contents of the alimentary canal in a separate receptacle from that in which portions of solid viscera are kept. Poison similarly found in urine, unless added from malice, also affords proof of absorption and excretion. If it were also found in the food or medicine preserved, but which had not been swallowed, this would be very strong corroborative evidence.

10. **If it was not a Poison, was it " noxious " ?** It is in answer to this question that attempts at the definition of a poison are so frequently made. (The reader is referred to pp 218 *et seq.*)

11. **Could the supposed Poisonous Substance exist naturally or be produced within the Body by any Process of Decomposition ?** This is a very important question, but one easily answered in the case of definite alkaloids, such as morphine, coniine, atropine, strychnine, etc. They could not, by any means known to experience or science, possibly be manufactured in the body from either its own tissues or from any food product, and their presence is incontestable proof of administration.

The discoveries by Panum, Bergmann and Schmiedeberg, Otto, Selmi, Brieger and many others that poisonous products could be extracted from putrid fluids and that these products resembled chemically and physiologically certain well-known alkaloids, led to the belief that they might play a part in certain intoxications from decomposed food. Selmi in particular emphasised their importance and called them ptomaines (πτῶμα, a corpse or carcass), and thus arose the concept of " ptomaine poisoning," a term in frequent use even at the present time. Later research has established the fact that a large number of ptomaines are

relatively non-toxic, only a few, such as neurin, muscarin, mydalein, approximating in toxicity the well-known alkaloids. These " cadaveric alkaloids " are not isolated by the Stas-Otto process.

Incidentally it is certain that the vast majority of cases of " ptomaine poisoning," so called, are due to highly complex poisons produced by or present in the bodies of certain bacteria, notably of the Salmonella group, and have nothing to do with ptomaines.

When metals are found in very minute quantities it again becomes a little difficult to swear that they could not have found their way in as constituents of foodstuffs, though they may not be natural constituents of the body. Copper, for example, is now regarded as being, in minute traces, an element essential to life, and minute traces of arsenic can be demonstrated in most materials, for arsenic is exceedingly widely distributed in nature ; it has been found in the " fur " of kettles and boilers, in river and well waters, in soil, in plants, and so on. Indeed, one of the difficulties in arsenic estimations is to obtain arsenic free reagents, and a second is to make proper allowance for the traces normally present in animal and plant tissues. It is, in fact, very dangerous to draw serious conclusions from the finding of minute amounts of arsenic in viscera unless there is also good physiological or pathological evidence of arsenic poisoning. Bamford[1] considered that in Egypt, at any rate, quantities of arsenic up to ·02 mg. per 100 g. of tissue must be regarded as " normal." The objection is, however, more theoretical than practical, for in all cases of a suspicious character the toxic metals are found in quantities too large to be accepted as arising from such sources, especially when the symptoms are taken into account.

12. **Could it have been present in the Reagents employed in Analysis ?** This, especially as regards arsenic and lead, is a very proper question for defending counsel to put ; the well-known fact that these metals are frequently contained in commercial chemicals (especially acids) should put an analytical chemist on his guard, and nothing but gross carelessness could account for their presence in reagents used for such delicate work as that now under consideration.

13. **Was it possibly due to a Preservative employed on the Body ?** It is this question which makes it so necessary to be careful about saving portions of the body for analysis without the use of antiseptics. Carbolic acid, corrosive sublimate and other mercurial preparations, chlorinated lime, etc., may all very easily confuse the result, and so may arsenic in paper used for wrapping up viscera (*vide* case on p. 262). If the body has been artificially embalmed or definitely injected for preservation, the fact will be obvious.

There are few reports in which answers to many of these questions will not be required ; and unless all of them are borne in mind by the operator at the time an analysis is undertaken, those which are omitted can never receive an answer, however important to the ends of justice that answer may ultimately become.

As a fitting illustration of the points raised in the foregoing discussion on poisoning in its medico-legal aspects, the report of a case that occurred in 1903,[2] with a few marginal comments, is inserted.

[1] " *Poisons : Their Isolation & Identification.*" Churchill, 1940, pp. 89–92.
[2] *R. v. Klosowski or Chapman.*

Severino Klosowski (36), alias George Chapman, was indicted for, and charged on the coroner's inquisition with, the wilful murder of Maud Marsh.[1]

For the defence it was submitted that the prosecution were not entitled to prove the deaths of any other women at previous dates, and which were alleged to have been brought about by the prisoner, and he was separately indicted for them. Counsel referred to *R.* v. *Winslow*[2] and *R.* v. *Oddy*, before the Privy Council in 1893, and *R.* v. *Makin*.[3] The Solicitor-General submitted that he was entitled to open the facts and give evidence of the death of other women with whom the prisoner had lived, and submitted that the case of Winslow had been overruled. He quoted *R.* v. *Gill*[4] and *R.* v. *Flemington*,[5] and also the case of Neil Cream. Mr. Justice Grantham ruled that the evidence was clearly admissible.

A legal point of evidence.

As the prisoner had lived under at least two names, evidence of identification was here of cardinal necessity. The evidence not only identified the man, but proved that he was well acquainted with drugs, which was a point of importance.

Evidence of identification of the prisoner and of intent.

The next evidence was the purchase by the prisoner in 1897 of an ounce of tartar emetic, and here the chemist who sold it had to show and did show that he took *all* the precautions required under the Sale of Poisons Act.

Evidence of compliance with Pharmacy Act; witness not bound to incriminate himself.

Evidence was then given to prove an alleged marriage of the prisoner to the victim, and also that in July 1902, the victim was in hospital with similar symptoms to those of which she died. [In October 1902, the fatal illness began, the main symptoms being vomiting and abdominal pain and excessive thirst; these recurred whenever she took drink prepared by the prisoner, and he prepared, or had access to, all her food.]

Dr. Targett gave evidence to the effect that her illness in July was attributed to peritonitis, and that she had fever (102° F.), and that therefore antimony could hardly be suspected to be the *whole* cause of her illness at that time, even if other circumstances had suggested it.

Evidence showing how easy it is to overlook a case of poisoning that does not end fatally.

Francis Gaspard Grapel: I practise at West Croydon, and have been the medical attendant to the Marsh family for some time. On Tuesday, October 21st, Mr. Marsh called on me, and in consequence of what he said I went about 3 or 4 p.m. to the "Crown"—I saw the prisoner and told him that I was a medical man from Croydon, and had come to see Dr. Stoker in consultation about his wife—he said there was a doctor already in attendance, and something about fifty others—I could not distinguish exactly what he did say—I asked to see Mrs. Marsh—I sat in the bar and then saw Dr. Stoker, and together we examined the deceased—her skin was sallow, jaundiced, and muddy in appearance, her tongue coated, her pulse fairly quick, her breathing shallow—she was in a semi-comatose condition—I examined her stomach—it was extremely tender to the touch—when I touched it she groaned and retched—I had a consultation with Dr. Stoker downstairs, and then saw Mrs. Marsh—before leaving the house I asked for and was shown some of the vomit—it was green—Dr. Stoker and I were of the opinion that she was suffering from some acute irritant poison, probably ptomaine—later on the suspicion crossed my mind that it was not ptomaine poisoning, but repeated doses of arsenic—I formed that opinion before there was a *post-mortem*—after I heard of her death I sent a telegram to Dr. Stoker.

Evidence showing the value of a consultation, and how suspicion may arise, and doubt of course of action when suspicion alone is present.

[1] C.C.C., March 1903.
[2] 8 Cox's Criminal Cases.
[3] Appeal Cases, 1894.
[4] 18 *Law Journal*, Magistrates' Cases, p. 66.
[5] 15 Cox, p. 403.

Troubles of a doctor when suspicion arises in his mind.

Cross-examined: It crossed my mind that it was arsenical poisoning on my way home—I did not go back and tell Dr. Stoker or send him any communication until after she was dead—bringing in a diagnosis of repeated doses of arsenic is tantamount to accusing someone of murder, and I had no proof whatsoever—I did not believe she was likely to die then—I was going to communicate with Dr. Stoker next day—the prisoner sent for the doctor—he did not put the slightest obstacle in my way of seeing the deceased—it did not strike me that he seemed anxious—I did not question him about the symptoms—he said she had been suffering from constipation—he did not tell me how she had been treated—I did not say more to him than I could help—on the Wednesday morning I told Mr. Marsh that I was going up to London as early as I could on that day to see Dr. Stoker, with the idea of having the excreta saved and examined—I did not examine Mrs. Marsh—I heard of the rabbit—I think it was Mrs. Marsh who told me that her daughter had been poisoned by a rabbit, and also the servant—I afterwards told the father that you did not get arsenic in a rabbit—it did not occur to me to investigate the story of the rabbit—I did not feel justified in at once telling the father what my suspicions were—even a doctor must have time to think about a case before he renders himself liable to anything legal—I did not take part in the *post-mortem*.

Evidence showing how a doctor may be, and is, imposed upon, but it should have aroused suspicion.

James Maurice Stoker: I practise at 221, New Kent Road. On October 10th, about 5 p.m., the prisoner called at my surgery, which is about half a mile from where he lives—he said he wanted a bottle of medicine for diarrhœa and vomiting—he led me to believe it was for his wife—he said she had been at Guy's, suffering from the same thing—he said she was not his wife, but she passed as such—I gave him a bottle of medicine; it was catechu, chalk, bismuth, and opium—the same evening about 10.30 I went to the "Crown"—I found the deceased in bed on the second floor—the prisoner went into the room with me—the deceased said she was suffering from diarrhœa and vomiting, and great pain in the stomach—I examined her stomach—there was great pain and tenderness all over the abdomen—I told her to continue taking the medicine I had sent, and said she was to have no solid food, but to go on a milk diet—I ordered her soda-water and milk, boiled milk, brandy, beef-tea, and ice—I ordered the ice to stop the sickness—she did not complain of any great thirst then—I went to see her the next day—the prisoner again went into the room with me—she was no better—the prisoner then said that she had been treated at Guy's, but they did not quite understand what was the matter with her there—the deceased told me that they had said she had peritonitis—the symptoms she complained of would be consistent with peritonitis—I saw her again next day (Sunday)—she was very much better—I changed the medicine and gave her bismuth, morphia, and ipecacuanha; that was for the soothing of the stomach—I saw her again on the 13th; she was then as bad as ever—she had diarrhœa and vomiting; I saw them both; they were mixed together; it was an ordinary yellow-brown mixture—I saw her again on the 14th; she was no better—about the fifth day I noticed she had spasms—they came on with great pain in the stomach; she got rigidity of the muscles of the leg; they passed off in about half a minute—they did not synchronise with the sickness—they came on independently—on one occasion she had two in about five minutes—I could not then form an opinion as to what caused them—I saw her again on the 15th: she was no better—I asked the prisoner if she was having the milk diet, and he led me to believe she was having all I ordered—there was no one else to ask—I was not there when anything was administered—she was very much worse on the 15th; and on that day I stopped all food through the mouth except the bismuth powders—she

Suspicious symptoms continuing.

could not even keep the medicine down—I ordered her to be fed by injections through the rectum—she was to have egg, milk, and beef-tea as a mixture—I could not then form any opinion as to what she was suffering from—I thought the symptoms might be those of gastro-enteritis, which is inflammation of the stomach and bowels—at that time I had not the slightest suspicion of any foul play—I suggested to the prisoner that she should be taken to the hospital, but the deceased objected and began to cry—I then suggested a nurse, but the next day, finding there was still no one there, I spoke to the prisoner about it—he said he had tried to get one, and that she would come on the following day—on this day I found that the deceased could not retain the bismuth powders—they were to allay the irritation of the mucous membranes of the stomach—I stopped them and advised her to be fed entirely with the injection—I got beef-tea suppositories and told the prisoner to give her everything iced—I do not know whether up to that time she had had any injections—on Friday I saw the nurse, Toon, for the first time—I gave her directions about the food and injections—I did not know if she knew anything about giving injections—the prisoner was there when I gave Toon the instructions—I thought Toon was carrying out my directions—I called again on Saturday, the 18th—I found the deceased very bad, vomiting and diarrhœa—I saw the vomit; it was slimy and green; the green would be due to the irritation of the stomach and gut—I do not think I visited her on the Sunday; I know I missed one day—the next time that I saw her after the Saturday I found her much weaker and with the same symptoms—I asked the nurse about the injections—she told me she did not even retain these—I told her she ought to reduce them to half the quantity to try if she could retain any liquid—the prisoner was there when I said that—I had no idea that it was the prisoner who was giving the injections—I was sent for to meet Dr. Grapel—the prisoner was not present—the deceased was very weak and semi-unconscious—I had some conversation with Mrs. Marsh on the landing about the death certificate on the Monday—I called on the Wednesday about 3 p.m. and then heard that the deceased had died at 12.30—a message had been sent to me, but I did not receive it, as I was out visiting—when I saw her on the Tuesday, I had no reason to anticipate that she would die so soon—on the Monday she was about as bad as she could be, and I could not say if she would get well—on the Wednesday I asked when the deceased had died—I was told—I then went out on the landing and had a conversation with the prisoner—I said I should like a "P.M." as I could not account for the cause of her dying—he said, "What use is it?"—I did not say anything about the certificate then—I went back into the room with him—Mrs. Marsh was there—I told her that I wanted a "P.M." as I could not account for the cause of death—she said "I must leave it to her husband"—I said that I did not know what was the cause of death, and I might be asked what had caused her death—the prisoner said that she had died from exhaustion—I asked what caused the exhaustion—he said, "Diarrhœa and vomiting"—I asked what caused the diarrhœa and vomiting, and he made no answer—I said I could not give a certificate for her—I would have to have a "P.M." or an inquest—I told the prisoner I only wanted a private *post-mortem* just to satisfy myself as to what caused the diarrhœa and vomiting—I then said I should have to make arrangements for the removal of the body to the mortuary, and I went to the proper authorities and the mortuary keeper—the body was removed early the following morning—on October 23rd I made a *post-mortem* examination with Dr. Cotter—I examined the liver, the kidneys, the lungs, and the ovaries—they were healthy—I examined the intestines and the stomach externally—I could not arrive at any opinion

Very proper medical procedure in suspicious cases.

Evidence of the right thing to do when suspicion is aroused.

Correct procedure in a suspicious *post-mortem*.

Incorrect procedure; compare statement with Bodmer's evidence p. 279.

Correct action when suspicion made a certainty, or nearly so.

Evidence rebutting suggestion that poison might have been in medicine.

as to the cause of death—I did not see anything to account for the symptoms causing death—I had taken two glass bottles with me when I went to make the examination—I had taken pains to see that they were chemically clean—I removed the stomach and its contents from the body without opening it—I tied it up and put it straight into one of the bottles—I also removed portions of the rectum and the liver, and put them into the other bottle—the deceased was not pregnant—there were no traces of pregnancy or any affection of the womb—I took the bottles away myself and sealed them up, and next day took them to Dr. Bodmer, of the Clinical Research Association—I got a communication from him in the evening, and then I had a consultation with Dr. Cotter, and as a result in the early morning of Saturday, the 25th, I communicated with the police—after the prisoner was arrested I saw these bottles of medicine (*produced*) which I had sent to the deceased—I make up my own medicines—I made these up—this small one had opium and water in it; that is a sedative; that was for injection to relieve the pain in the rectum—I cannot fix the date that I ordered that—this other bottle, I should say, had bicarbonate of soda and prussic acid in it—I did not put antimony or tartar emetic into any of them—I do not keep any antimony in my surgery—I had none at this time—I have had none for ten years—I do not know if antimony now is an accepted medicine; I do not know much about it I never use it—I keep a preparation of arsenic—I did not put any arsenic in the medicine—I think these are some of the bismuth powders I prescribed (*produced*)—I had some of it remaining in my surgery after the death of the deceased—I handed it to Inspector Godley with a view to it being analysed—I was present at the *post-mortem* made by Dr. Stevenson.

Cross-examination to destroy witness's reliability, but he very correctly assumed no special knowledge. Note how he was questioned on his knowledge of drugs.

Cross-examined: I have been in practice ten years in London, and have had a good many cases of ptomaine poisoning, but not of arsenical or antimonial poisoning—I had never had a case where poison has been deliberately administered—this is my first experience of a case of this kind, and I hope it will be the last—I know that tartar emetic is in the Pharmacopœia as a medicine; I have never had occasion to employ it—I have never had any practical experience of the tartarisation of antimony—I do not think I should recognise it if I came across it in a *post-mortem*—if antimony was present in the deceased's stomach when I took it out and placed it in the glass jar I should not recognise it—I do not put myself forward as having any special knowledge on that subject—I should not have analysed the deceased's vomit myself—I should not have tried to trace the presence of antimony—the prisoner did not in any way object to my making an examination of the vomit or the excreta—I could have taken it away for examination; the vomit was only unusual because it was green, but not more so than you would see if the patient was suffering from a bilious attack—I have constantly seen vomit as green as that—I did not see any blood in it; it may have been there when I did not see the vomit—I only saw it on two occasions—I only saw the fæcal matter once—I saw no blood then—I prescribed altogether six five-grain bismuth powders—I am not aware that bismuth gives rise when tasted to the same burning sensation in the throat as is alleged with regard to antimony—I do not know if it gives rise to a metallic taste—I have often taken bismuth, but never tasted it—I have taken a piece on my finger—I have never had a case of poisoning from bismuth—I know there are such cases, but you want an enormous quantity—you can get diarrhœa and vomiting from an overdose of bismuth—you might get spasms in the arms and legs—I do not know if you would get inflammation of the throat, windpipe, and gullet—I prescribed the ipecacuanha in three minim doses—I should say about sixty minims

would give rise to a sense of sickness—I have never known ten or twelve minims doing so—the ordinary dose goes up to ten—some people are more sensitive than others—I did not keep any record of the medicine I prescribed—I never do so except with a patient coming into the surgery—I do not enter in a book the medicines I send out—I put down the visits I make—it is not unusual to have no record—I am almost certain that my memory is accurate as to what I gave the deceased—the beef enules came from Burroughs & Wellcome—the patient generally gets the nurse; the prisoner had no objection to having one—I did not know what Toon's experience was—I did not think it necessary to satisfy myself that she could carry out my instructions—I thought the prisoner would get a good nurse because he knew what had to be done—I should not as a rule witness the injection—the prisoner asked me to call and see the deceased—I did not suggest to him during the last twelve days that he should call in another doctor—I did not know Dr. Grapel—the prisoner was not present at our consultations—I was in consultation with three or four other medical men in connection with that matter with the prisoner's consent and approval—he paid the fees—I was always under the impression that any suggestion of mine with regard to food was carried out—he appeared to be kind and solicitous to his wife—she appeared to be fond of him—I had never the slightest idea of anything being wrong—the vomiting and diarrhœa were consistent with my experiences of gastro-enteritis—I should have been perfectly ready to give a certificate to that effect.

Value of records of all cases.

Re-examined: I did not think of submitting the vomit to chemical analysis because suspicion was entirely eliminated from my mind—I had no idea of any irritant poison; it would not be possible for me to recognise antimony when examining a body—it would have to be submitted to a chemical analysis or to somebody with more experience that I have—I have never seen a case of poisoning by bismuth; I have read of one—there has not been a case in recent years—the number of grains in a case I read of was very great; at that time bismuth had arsenic in it—the bismuth in my surgery was found perfectly pure—a five-grain dose of bismuth is a medium one—I was prescribing it with a view to allay the vomiting—it is a well-known remedy for doing that—I have never heard of these small doses causing vomiting or irritation—it always had the opposite tendency—I use about half a pound in a fortnight—I did not know whether my instructions as to diet were carried out or not.

Doctor not sufficiently suspicious in such a case.

By the Court: I know Bessie Taylor died at the "Monument"—her symptoms were something similar to those of Maud Marsh—even that did not make me think at first that I ought to have further inquiry.

Richard Bodmer: I am a Fellow of the Institute of Chemists and Public Analyst for the Borough of Bermondsey—I am consulting chemist to the Clinical Research Association, 1, Southwark Street—I received two sealed jars from Dr. Stoker—one of them contained a human stomach and a small piece of human liver—the other contained a part of the lower bowel and some pieces of liver—I had a conversation with Dr. Stoker, and in consequence I applied some tests in order to discover whether there was any arsenic present—the stomach was tied at both ends, and on opening it I found its contents were from about one and a half to one and three-quarter ounces of a yellow gruel-like fluid—I applied Riensch's test to a small portion of it; that is a well-known test for arsenic—I discovered arsenic was present—some slips of copper are used in the test; they became a purple colour, indicating the probable presence of antimony in addition to arsenic—I communicated what I found to Dr. Stoker—on Monday, October 27th, Inspector Godley saw me, and in consequence I subjected another portion of the

Evidence of identification of jars, etc.

Mixing of solid viscera with pieces of gut very careless.

contents of the stomach to Marsh's test, which is also a test for arsenic and antimony—I discovered both present by that test, and also found that there was far more antimony present than arsenic—I did not open the second jar—I replaced the stomach in the first one, and a part of it which I used in my tests I placed in a perfectly clean glass-stoppered bottle, and on October 28th I handed all three jars to the coroner's officer.

Cross-examination of expert to try to find incorrect inferences.

Cross-examined: The violet deposit suggested antimony to me—it is not always a conclusive proof of its existence—other substances will hardly produce the same colour, but something which might be taken for it—I have not read a report of Pritchard's case in 1865—I cannot at present remember any other substance which would produce the same violet colour—sometimes arsenic will come out and almost look like antimony—I do not only rely on the colour of the copper—I subjected the copper to a cleansing process before I used it.

Not medical, but important, evidence *re* identity and safe custody of objects for analysis.

Joseph Henry Vaughan Marks: I live at 31, Gaywood Street, and am coroner's officer for Southwark—on October 25th I got some information and went to the police-station, and an inquest was opened on the body of Maud Marsh on the 28th—that evening by the coroner's order I went to Mr. Bodmer and received from him three jars—I took them to my house and kept them there till October 30th, when I gave them in the same state to Dr. Stevenson.

Evidence of the expert on the *post-mortem* and analysis, ranging even to the first doctor's drugs, illustrates the wide and accurate knowledge required of a toxicologist. Note his cross-examination.

Thomas Stevenson, M.D.: I am one of the official analysts to the Home Office, and also Lecturer on Forensic Medicine at Guy's Hospital—I have had experience in analysis, particularly with reference to poisons, and have acted for the Home Office for thirty-one years—On October 30th I attended St. George's Mortuary to make a *post-mortem* examination on the body of Maud Marsh—Dr. Freyberger, Dr. Stoker, and Dr. Cotter were there—there had been a previous *post-mortem*—the body had been dead fully eight days, but there was not much decomposition, much less than I should expect in a body so long dead, considering the time and season—the scalp covering the skull was dry; that indicated that there was little fluid in the tissues—the skull and brain were normal—there was no hæmorrhage or disease in the brain—the spinal cord was normal, and no sign of disease there—the tongue was yellow, coated, and swollen—the air passages to the lungs were quite clear, and the lungs free from disease—there was a good deal of fat about the heart, but that would not have affected her health much, unless it had gone much further—it had invaded the muscles to the extent of about one-third—the mesenteric glands were much swollen—the stomach had been taken away, but it was given to me by the coroner's officer before the end of the *post-mortem*—the blood-vessels of the bowels were unusually red and injected with blood, but not to a very marked extent—the mucous membrane of the bowels was swollen and slimy, and was in the condition which we generally know as subacute enteritis, which is inflammation of the membrane lining the bowels—there was a good deal of liquid in the bowels, but only a little semi-solid fæcal matter, which was about the sigmoid flexure of the colon—one of the glands showed that she had probably been a person subjected to habitual constipation—the whole of the rectum had been removed—I found no ulceration of the bowels—I examined the pancreas, the spleen, and the kidneys; they were all sound and healthy—the liver had been detached, but it was in the abdomen—a small portion had been removed—it was rather dry and greasy but there was no condition which would affect her health materially—I examined the womb and ovaries—they were perfectly normal—she had never apparently borne a child, nor were there any signs that she had been far advanced in pregnancy—menstruation was just upon ceasing—I found no evidence of any natural disease

which would account for her death—I suspected that she had died from some form of irritant poison, which had set up enteritis—I had heard of the question of arsenical poisoning, but I came to the conclusion on making the examination that it was not arsenic, but some other metallic poison—I then removed the brain, some blood from the cavity of the chest, the spleen, the gall bladder, which was full of bile, the liver, the kidneys, the contents of the bowels, the bowels themselves, and also some blood from the abdominal cavity—they were all rather light in weight—the drain on the fluid caused by the vomiting would account for that in a great measure—on the 31st I examined the stomach from the jar—there were signs of putrefaction externally and internally, it was pink and injected with blood—the blood-vessels were prominent and redder than usual—internally it was coated with a good deal of yellow slimy mucus, which became an orange colour at the bowel end—I did not find any ulcers or loss of substance—I examined the contents in the stomach and portion of the liver and rectum in the jars—I made an analysis of various parts of the body—every portion of the body which I examined had antimony in it—I found antimony in the stomach and its contents, in the bowels and their contents, in the liver, bile, spleen, kidneys, the fluid which I took from the abdominal cavity, in the blood from the cavity of the chest, and in the brain—I made tests for arsenical poison—I found traces of arsenic in a small quantity, and I formed the opinion that death had not resulted from it—arsenic is sometimes found in antimony when it is impure—I came to the conclusion that death was caused by poisoning with antimony in a soluble form—that was tartar emetic or metallic antimony—that is one of the scheduled poisons—I did not find any bismuth there, but the tests for bismuth are not so complete—if there was any it must have been infinitesimal—I have never heard in late years of a case where bismuth has caused such symptoms as these, or caused death—bismuth is now purified from arsenic and other impurities—five grains is an ordinary dose—in the cases I have heard of where death was caused by bismuth I think that 120 grains must have been taken—I found no trace of impure bismuth in this case—I found 0·23 grain of metallic antimony in the contents of the stomach, 5·99 grains in the contents of the bowels—that indicated to me that there must have been a large dose of antimony given within a few hours of death, as it is soluble in water—it had not been got rid of by purging or vomiting—in the liver I found 0·71 grain of metallic antimony, in the kidneys 0·14 grain, in the brain 0·17 grain, in all 7·24 grains, which is 7¼, the bulk of which was in the bowel—I deduce from that that there was a good deal more antimony in the body—antimony can be made soluble in the form of tartar emetic or emetic tartar, which is a white powder, soluble in water—it does not change the appearance of the water—emetic tartar is not altogether antimony—7·24 grains of metallic antimony would represent 20·12 grains of tartar emetic—the proportion is roughly three to one—I did not calculate the amount of tartar emetic in the whole body, but from my experience I should put it at between twenty-five and thirty grains—when tartar emetic or antimony is administered as a rule the greater part of it is very quickly ejected—purging removes it—the effect of the poison itself generally takes a very considerable time before it causes death—death has occurred in many cases where it is given in repeated moderate doses—vomiting and purging makes people waste away—it produces gastro-enteritis, and they also appear to die from failure of the heart—antimony depresses the circulation—it quickens the pulse, but gives it a very feeble power—two grains of tartar emetic has killed, but that is not ordinary—I should put the ordinary fatal dose at probably fifteen grains—others put it at ten—even that might not be fatal if the greater portion of it is vomited—people

Close examination on symptoms of the alleged poison.

have taken tartar emetic in much larger quantities, and have recovered where it has been quickly vomited—I am of opinion if two or three grains were given repeatedly to a healthy person that it would eventually cause death—when doses of antimony are given from time to time the symptoms are great depression, profuse perspiration, followed by nausea and vomiting—purging is set up with pain in the abdomen, and usually after a time there is a burning or metallic sensation in the throat and stomach—there is a great thirst—spasms are quite common, and patients fall sometimes into a comatose or semi-comatose state—they are generally very pallid, and sometimes they get jaundiced, and dark under the eyes, and thin and worn—it is sometimes the appearance, apart from other symptoms, which indicates that the patient is approaching death—in the case of Mr. Braywood it was his appearance which excited the suspicion that he would die—Sir William Gull and others who saw him, although he was apparently going on well, thought afterwards he could not get on so well—that was a case of poisoning by tartar emetic—if tartar emetic is taken in a strong solution it has a somewhat metallic but sweetish taste, but when taken diluted it does not have much taste—it can be covered up by food or medicine—people take antimony wine, which is sherry with antimony in it, and do not know that anything is wrong—if doses had been going on for some time so as to set up irritation of the mucous membrane, that would set up the burning feeling in the throat—antimony can be dissolved and given by injections, or put into injections—that would be very dangerous: it would be quickly absorbed into the rectum and then into the body—the vomit in the case of poison by antimony would be at first the contents of the stomach, and then it would become green or yellow—I got a great number of bottles from Sergeant Kemp—I examined them—there was antimony or arsenic in one of them—some of them had contained photographic chemicals—I examined the bismuth powders found in the room, proved to have come from Dr. Stoker—they were free from antimony and arsenic—I also examined the bismuth from Dr. Stoker's surgery—it was pure and a very good specimen—these two bottles which contained medicine had no trace of antimony or arsenic—this other one had two or three drops at the bottom of it—I do not know if it had been washed out—I found bismuth and antimony in it—I should say there was quite as much antimony as bismuth in it—there was enough antimony to give several dull doses—emetic tartar can be dissolved in water so as not to be apparent, and then could be mixed with a bismuth preparation—tartar emetic is also soluble in brandy—brandy of ordinary strength will take up about two grains to the ounce—a tablespoonful of such medicine would be a full emetic dose—it is much more soluble in brandy and water than in plain brandy—these two bottles contain brandy and water, two parts water to one part brandy—there is tartar emetic in one of them—this one, which contains about an ounce, is probably a fatal dose—I could find nothing to account for death except poison by antimony—antimony might be given to produce perspiration and for bad colds, but only from one twenty-fourth to one twenty-sixth of a grain to a dose—half a grain to a grain would be an emetic—death would not ensue from one dose of that strength or cause great pain—if vomited, it would not produce diarrhœa—all the signs I found at the *post-mortem* would not be caused by such doses as that.

Doctor's medicines analysed.

No other cause for death.

By the Court: When I said I could find nothing to account for death except poisoning by antimony, I meant antimony administered for the purpose of poisoning.

Cross-examined: There was not enough antimony left in this bottle for a fatal dose—if the bottle had been full of the same preparation of bismuth, antimony, and water, I think there would

have been sufficient for a fatal dose, but I am not positive—it contained six doses—each dose of itself would not be a fatal one, but I cannot say what would be the effect of six successive doses—I should expect a person to be very ill after taking them—I examined the syringes—one of them is covered with an insoluble preparation of antimony, which is used in its manufacture—apart from that I do not find any trace of antimony on either of them—I heard that they were used for the injecting of liquid beef-tea, but also that they were soaked and washed afterwards—the immediate cause of death was subacute gastritis—I have had to do with a good many cases, but I never saw such extensive gastro-enteritis from ptomaine poisoning—it generally produces more inflammation of the bowels than of the stomach—in this case the stomach was worse than the bowels—persons who have died from ptomaine poisoning suffer from subacute enteritis—it is a question of degree—the fat which I noticed round the heart might point to old alcoholism, but fatty degeneration of the heart is well known in poisoning—the deceased was a very young woman to have had a fatty heart from alcoholism—tartar emetic is sometimes used as a check to drinking propensities—it nauseates the patients so that they cannot indulge in drink for the time being—in former times bismuth contained impurities which, apart from the bismuth itself, gave rise to arsenical poisoning; but at the present day bismuth is cleansed from those impurities, and is not itself poisonous—there have been cases on record of bismuth poisoning where the dose was only two drachms—I have edited Taylor's "Medical Jurisprudence"—a metallic taste, vomiting, and purging, spasms in the arms and legs, which occurred in a case of bismuth poisoning, would coincide with a case of poisoning by antimony; but in a paragraph in my edition of Taylor I think that this is explained—my reason for saying that I did not attribute the poisoning to arsenic was because the quantity of arsenic was very small, and it was present only in the contents of the stomach and bowel, and in the liver, but I could not detect any in the more remote organs, and if a person died from arsenical poisoning I should expect to find arsenic generally distributed through the body—there was not sufficient arsenic to make me attach any importance to it—I ascertain its presence in various ways—the colour of the copper foil in Reinsch's test is very significant—arsenic, antimony, bismuth and mercury give very much the same colour, but none of them give the particular effect of antimony, so far as I know, and, of course, one does not rely on that one simple test alone—my tests are absolute, and not only probable—I discovered some orange sulphide, which was soluble in hydrochloric acid, and separating it and treating it by Marsh's process, it was proved over and over again beyond all doubt to be antimony—that colour is not common to other mineral or vegetable poisons—putrefaction had only just commenced in the body of the deceased at the time of her death—there was only one day when the temperature rose about 50 degrees—I keep a record—I was engaged in the Bravo case, which was a case of poisoning by tartar emetic—I did not see the vomit in that case, but probably it contained blood—the purge contained blood, but it is not invariably present in the vomit and purge in cases of antimonial poisoning—I have not personally had a case where a person who died of antimonial poisoning vomited or purged blood—Bravo passed blood the same night that he had the antimony—I was at the inquest in that case—there are cases where there has been no vomiting of blood.

Enema syringes examined.

Caution of quotations from books.

Cross-examination on tests used.

Cross-examination on symptoms of antimony poisoning.

Re-examined: The question of blood in the stool would be according to the amount of the irritation, and in the fæces the blood might not appear as blood, but as a black stuff—to an unskilled person it might not appear to be blood—if the motion was black it would probably indicate that there was blood—if I as a medical man wished to know whether there was metallic poison,

Re-examination on other possible cause of death.

I should look to see if there was blood in the faecal matter—these glass tubes contain antimony in a sulphide form which I took from the deceased's body—here are some in the metallic form—the shining portion is the sulphide from the bowel—all the bismuth coming from Dr. Stoker was pure, and did not contain arsenic—I have no reason to think that the deceased was addicted to drink—I never heard it suggested until now—there was nothing in the fatty degeneration of the heart that would cause death—I daresay I have a much fatter heart than she had—it would not have produced vomiting or gastro-enteritis—the poisoning could not possibly bring about the production of antimony by any internal process—if the syringes were soaked in water, that would get rid of any traces of antimony—there would be no difficulty in mixing the antimony with bismuth if you dissolve it in water first—you could also put it into the bismuth without dissolving it.

By Mr. Elliott: Bismuth taken by the patient produces black motions.

Shows a witness answering questions by jurymen more or less to the point.

By the Jury: There was less antimony in the rectum than higher up in the bowel—I cannot say if the whole of the last dose of antimony was due to rectal administration, but I think that she must have had during the last few hours of her life some given by the mouth—if the brandy was pure it would not take up enough antimony to give it any extra taste; but I should not like to take a mouthful of this brandy and water and antimony.

This finished the actual case of Maud Marsh, with the poisoning of whom the prisoner was charged, but evidence was then taken with regard to another woman with whom the prisoner had lived previously. The evidence here again, from a medical point of view, is most instructive.

Proper identification in exhumation

Thomas Stevenson (*Re-examined*): I attended at St. Patrick's Cemetery, Leytonstone, on December 9th, 1902, and examined the body in the coffin bearing the name-plate of Mary Isabella Chapman, who died on December 25th, 1897—I saw the lid removed—the body was altogether remarkable—the face and head were those of a woman who might have been coffined that day from the appearance—even the eyes were unruptured, a very unusual circumstance—there was not the least difficulty in recognising her—the muscles had a fresh appearance—all the parts of the body cut rather leathery, like shoe-leather, and of course were drier than in a fresh body—

Again, full knowledge of pathology required.

all the parts of the body except the brain were preserved—the stomach was unusually pink externally—that was from the blood in the vessels being more than usually good—its inner coat was of a peculiar cinnabar red colour, and towards the bowel end there was a patch of black blood which had been effused—there was no sign of perforation or ulceration—there was no loss of substance in the mucous membrane—towards the bowel end there were some old scars of years standing—the bowels were not ruptured—the tube was intact—internally the bowel had the same red colour as the stomach—there was no ulceration—the liver was pale, but firm in texture and fairly normal—the spleen, the kidneys, the bladder, the heart, and the lungs were all normal—there was no sign of phthisis; that generally indicates disease of the lungs—the cause of death was gastro-enteritis—there was no other cause—there was nothing to indicate that the woman had been a confirmed drunkard—if she had drunk it had not produced any serious injury to the kidneys or liver—the inflammation which I found in the stomach was not attributed to alcohol—I removed the stomach, the bowels, liver, kidneys, spleen, lungs, heart, brain, and some of the muscles, and submitted them all to analysis, except the lungs—

Preservative action of antimony.

they all contained antimony—it had permeated to the muscle of the thigh—in the bowels I found 0·41 grain of metallic antimony, in the liver 0·87 grain, in the kidneys 0·06, and the stomach 0·03, which

makes altogether 1·37—that would represent as emetic tartar 3·83 grains—there was more in her liver than I found in Maud Marsh's—the quantity points to a large amount of antimony having been absorbed into the body, and would indicate a considerable dose having been taken some hours before death or the continuous administration of small doses—the purging and vomiting would get rid of a good deal of antimony—I came to the conclusion that the cause of death was poisoning by antimony, and I attribute the preservation of the body to the antimony—it has not been thoroughly recognised that preservation is one of the effects of antimony, but it has been found in previous cases to be a preservative—the fact of antimony being found in the muscles would not indicate that doses of antimony have been going on for some time, because I think it would quickly pass to every vascular part of the body; evidently the body had not been touched by water, the coffin and its contents being well preserved.

Attempts to suggest that burial (*vide* Chapter V) influenced the condition of the body, and not antimony.

Cross-examined: The condition of preservation in a measure depends on the surroundings of the body quite apart from anything internal—it was an elm coffin—the grave was eighteen feet deep—the depth of a grave to some extent helps to preserve a body, but if this body had begun to decay at the time it was buried, the depth of the grave would not have retarded it—the air generally reaches a body before it is buried—this soil was very dry clay and loam, which would assist preservation—it would take a few years for rain to get down eighteen feet—the grave was not a brick one—there were seven other coffins above; this one was at the bottom—the body was almost lifelike—bodies buried in lead coffins, when opened years afterwards, have been found to be preserved to a wonderful degree—in those cases the air had been excluded—a wooden coffin would not be hermetically sealed—the other bodies removed from this grave had a fearful smell—we did not open the other coffins; they were reverently put aside, and a tarpaulin put over them—the whole of them had been buried within a month—I did not analyse the lungs, because I was told the woman had died from phthisis, but when I found no traces I put them aside in case questions were asked—if I had not known the history of the woman, but was told that a certificate of death from phthisis had been given, I might possibly have found that consistent with her condition—when people die from phthisis there is generally great emaciation.

By the Jury: I am of opinion that antimony given in gradual doses for a long time would be more likely to preserve the body than a sudden dose—it would get more into the system.

R. Bodmer (*Re-examined*): I took part with Dr. Stevenson in making an analysis of this body—I have heard his evidence, and I agree with him.

Shows how even with four consultants no suspicion arose.

J. M. Stoker (*Re-examined*): I was called to the "Monument" on January 1st, 1901—previous to that Bessie Taylor had called on me and asked for some medicine—I then attended her—I visited "Mrs. Chapman" almost daily from January 1st to February 13th, when she died—when I first called she was in bed; she had vomiting, diarrhœa, and pains in the stomach, which was very tender—the vomit was green—I cannot recollect if I saw her vomiting—I prescribed for her—she used to get better and then go back again—I suggested another doctor being called in—I had three separate consultations with three other doctors—one was Dr. Sunderland—he is a specialist in the diseases of women—he only saw her once—I was under the impression that "Mrs. Chapman" was suffering from some womb trouble—I do not recollect if Dr. Sunderland suggested any alteration in the treatment—she did not make any improvement—I then suggested another doctor—somebody in the house suggested Dr. Thorpe, of Southwark Bridge Road—he and I

examined "Mrs. Chapman" together—he said he thought she was suffering from a severe form of hysteria—I then got Dr. Cotter—we examined the patient together—he thought she was suffering from some cancerous disease of the stomach or intestines—in consequence I sent a portion of her vomit to the Clinical Research Association, with directions to see if there was any trace of cancer—that would be a microscopical examination—they found no trace—the constant vomiting and diarrhœa continued more or less during the whole time I was there—I remember one evening going in and finding her playing the piano—I cannot recollect the date—she appeared very much better, and in consequence I said I would not call back again unless I was sent for—I do not recollect if I had any conversation with the prisoner on that day—I was sent for the next day—I found her worse than ever—I was with her the day before she died—she was very bad then with the same symptoms—I do not recollect whether on that day I thought she was dying or not—next day I heard of her death—I was asked to give a certificate, which I did, giving the cause of death as intestinal obstruction, vomiting and exhaustion—intestinal obstruction would cause vomiting and exhaustion—she was suffering from vomiting and ordinary stoppage when she came to my surgery—diarrhœa would follow when the stoppage was cleared—I did not put the particulars in the certificate, "G. Chapman, widower of the deceased"—I thought the prisoner was married to the woman—I never had such a thing as antimony at this period, and I never prescribed it.

Illustrates the case of deceiving a doctor.

Cross-examined: I had seen Mrs. Stevens before—it was at her recommendation that I went to the Chapmans—up to that time I knew nothing of them—at first I regarded the case as one of constipation, and I directed my treatment with a view to removing that—I attended her at her home for excessive diarrhœa, so the stoppage must have given way—I think that she came to my surgery twice—I do not remember what I gave her, most likely a dose of salts—I next saw her at her home on January 1st, when I treated her for diarrhœa and vomiting—I do not know what I gave her then—I saw the prisoner—I do not remember if when I suggested to him that I should like further advice it was within the month—he at once agreed—I believe that Dr. Sunderland came to the conclusion that she was suffering from some uterine trouble—I have no record of it—I have not seen him since—the prisoner saw Dr. Sunderland when he came, and asked him what was the matter with his wife—I do not remember that he was dissatisfied with our opinion—he was willing to have a third doctor—he paid the fees—I consulted with Dr. Thorpe—I think he told the prisoner that the woman had hysteria—he accepted that opinion, as he had accepted Dr. Sunderland's—it was my suggestion that a fourth doctor was called in, because the woman was getting no better—the prisoner agreed to that—I do not know what fee he paid in each case—he did not grumble—Dr. Cotter said it was some cancerous disease of the stomach or intestines—I think Dr. Bodmer examined the vomit that I sent—Dr. Cotter's opinion was not sustained—I do not think the report was told the prisoner—I do not think the patient lived long after that—I had many opinions—I have no record of my treatment—if the specialist had suggested an alteration in the medicine I certainly should have made it.

Difficulties of diagnosis of poisoning.

Re-examined: None of us suspected poison.

By the Court: As far as I can tell, she was cured of constipation—you can get vomiting with hysteria, and you can imagine a lot when you have hysteria—I think Dr. Thorpe thought the woman was imagining—it did not occur to me that she was not suffering—constipation was the primary cause—the vomiting and exhaustion had caused her death—it would probably have been wise to have

had a *post-mortem* before giving the certificate, as all the doctors were evidently wrong—I have never known a case where four doctors gave four different opinions, and when the patient died, still there was no *post-mortem*.

Dr. Stevenson (*Re-examined*): On November 22nd, 1902, I was at Lymm Churchyard, Cheshire, and I saw Bessie Taylor's coffin taken from the grave—it had a plate on it, "Bessie Chapman, died February 13th, 1901, aged 36 years"—the body was covered with a mouldy growth, but otherwise was fresh—there was no putrefaction and no odour—the tissues were dry—the muscles had a red and freshish appearance—there was a fæcal odour in the abdomen, but no putrefactive odour—although the features had mould on them one could follow the shape and general contour—the breast was shrunken, and the whole body dry—generally when the bodies decompose they become wet and slimy—this one was extremely well preserved except for the superficial skin—I made an examination of the various organs—on the base of the right lung I found some old adhesions from old pleurisy—the lungs were shrunken and dry, but otherwise healthy and free from deposits or cavities—adhesions are quite common in people of good health in middle life and after—the heart and its valves were healthy—the stomach was empty, but its vessels were filled with dark blood to an unusual extent—on the inner surface of the gullet end of the stomach there was a patch about four inches in diameter of a cinnabar red colour which denoted gastritis—there was no ulceration or perforation or any loss of substance in the mucous membrane of the stomach—the cinnabar red colour extended more or less through the bowels, indicating enteritis—the inner surface of the bowel was coated with a yellow paint-like stuff, which was sulphide of antimony—the pancreas, spleen, kidneys, and liver were all shrunken by time, but otherwise normal—the womb, ovaries, appendages, and bladder were quite normal—I found no trace of cancer or uterine trouble—it was a good deal decomposed—there was no sign of hæmorrhage, or any recognisable disease—I found no intestinal obstruction—I formed the opinion that she had died from gastro-enteritis, which was due to some irritant poison—I removed the stomach, bowels, liver, spleen, kidneys, heart, brain, and lungs, and subjected them all to analysis and examination—the analysis showed that antimony was present in all those parts—there was no other poison—in the stomach there was 0·12 grain of metallic antimony, in the bowel 8·43 grains, in the liver 1·64 grains, in the kidneys 0·30 grain, making a total of 10·49 grains, which equals $10\frac{1}{2}$—that represents of tartar emetic in the stomach 0·32 grain, in the bowel 23·43 grains, in the liver 4·55 grains, in the kidneys 0·82 grain, making in all 29·12 grains—I cannot find any recorded case of such a quantity having been found in the bowel after death—it suggests that she had some large dose not long before her death—I examined the earth about the coffin, but found no poison.

Vide discussion on decomposition: "It is never too late to do a *post-mortem*."

Cross-examined: The woman had been buried about twenty-one months—Isabella Spink had been interred practically five years—neither body was putrid—Taylor was covered with ordinary vegetable fungi—there were conditions about the body that I identified with the case of Spink—I compared Taylor's features with a photograph which I was told was hers, and I could recognise the general contour—the nose and cheeks had preserved their shape—I could not distinguish the eyes. There had been a change in her which was more remarkable than in the case of Spink, where there had been practically none—I think Taylor's body contained more antimony than Spink's—given the same conditions as far as the coffin and grave were concerned, I should have expected to find that a woman who had only been buried twenty months, and had more antimony

Preservative effects of antimony.

in her body, to be less subject to change than a woman who had been buried five years and had less antimony—Taylor's coffin was a dry elm one, and, as far as I could judge, the body had not been contaminated by contact with the soil, which was very dry and sandy loam—putrefaction generally begins through the nose, mouth, and anus, and spreads outwards—there was none of that in either body—the superficial decomposition of the body was due to the growth of mould—the presence of antimony does not prevent the growth of moulds; in fact, they will grow in a strong solution of tartar emetic.

Verdict, "Guilty"; sentence, death.

CHAPTER XVII

CLASSIFICATION OF POISONS AND POISONING BY (GROUPS OF) INDIVIDUAL POISONS

There is as much difficulty in classifying poisons on any satisfactory and scientific plan as there is in defining a poison. They may for various purposes be classified in many ways, according to their origin, for instance, from the animal, vegetable, or mineral kingdom, or according to their mode of entrance to the body, or according to their physical properties, *i.e.*, gaseous, liquid, or solid: but to each and all of these there are objections of greater or less magnitude.

There is one method which would be unexceptionable if it were only practicable, and that is to classify them according to their action on the body. Of this method a sketch containing a few substances will be found on p. 230. If an attempt were made to complete the list by adding to it even all the well-recognised poisons, nothing but confusion would result, for so many of them act in more than one direction, and not only so, but sometimes in one direction and sometimes in another.

If science fails us, the only principle left is convenience, and here some assistance is obtainable by grouping certain poisons for descriptive and other purposes. The following groups have considerable convenience to recommend them, and there is also some connection between the members of each group, if not between the groups themselves: —

1. Corrosives, strong acids, and alkalies (including phenols).
2. Poisons derived from metals and their salts.
3. Gaseous poisons.
4. Poisons derived from the non-metallic elements.
5. Anæsthetising agents, sedatives, and antipyretics of artificial origin.
6. Artificial organic substances not in group five.
7. Poisonous substances of direct origin from the living vegetable kingdom.
8. Poisonous substances of animal origin.
9. Food poisons.
10. Miscellaneous poisons—mechanical, such as glass, coffin dust: patent medicines, etc.

Group 1. THE CORROSIVE POISONS

Of this class the typical members are the concentrated mineral acids and caustic alkalies, and so long as these only are considered the group is a very natural one, but there are many objections to considering it a scientific one all the same. For instance, nitric and hydrochloric acids may by their volatility act as simple pulmonary irritants,

while by dilution sulphuric acid may act as an irritant only. Again, oxalic and carbolic acids should at times be included in the group, for they frequently kill by purely local action, but, on the other hand, they often do not corrode, but kill by their poisonous effects after absorption. The group, then, must be considered to consist typically of the strong mineral acids and alkalies and to shade off through the more dilute and less powerful acids down to the irritants.

The total number of deaths due to the ingestion of the corrosive poisons is much greater than one would expect in view of their violent and painful action and obvious physical characters.

The main principle connecting the several members of the class is that they *may* cause death by purely local destructive action, combined with shock and collapse produced by intense stimulation of sensory nerves, dehydration due to vomiting and diarrhœa and alteration in the alkali reserve of the body tissues. In addition to the local effects certain of the corrosive poisons such as Phenol and Oxalic acid exert a profound effect on the central nervous system after absorption.

Test for Free Mineral Acids

The presence of a free acid is not proved by identification of the acid radicle; it is necessary to demonstrate that the radicle is really present as an acid and not as a metallic salt. This can sometimes be done merely by testing a watery extract with a suitable indicator (congo-red, methyl-orange, thymol-blue, etc.) sensitive only to such high concentrations of hydrogen ions as are produced by mineral acids or unusually concentrated solutions of organic acids. Litmus is, of course, inadmissible, since it is affected by organic acids and even by carbonic acid, and even congo-red reacts with concentrated vegetable acids. More conclusive evidence may be obtained by evaporating a little of the material with freshly made Gunzberg's reagent; the presence of mineral acid is shown by the appearance of a red or red-rimmed residue.

The presence of free mineral acid having been proved, the identity of the acid may be established by the ordinary tests used in analytical chemistry.

If free sulphuric acid, mixed with other acids, is suspected, the aqueous extract should be evaporated on a water bath to remove volatile acids, and the residue extracted with equal parts of alcohol and ether. This mixture extracts free sulphuric and phosphoric acids from admixed salts. It is stated that sulphuric acid itself is seldom found, owing to its reaction with phosphates in the organism; but the presence of phosphoric acid in the alcohol-ether extract would indicate either the ingestion of free sulphuric acid or free phosphoric acid.

Free hydrochloric acid exists normally in the stomach. Hence examination of the stomach and its contents by these qualitative tests cannot provide evidence of any value and a quantitative analysis is necessary. By titration of a measured volume of the material with standard sodium hydroxide solution using methyl-orange or thymol blue as indicator, the concentration of free acid can be determined; this will not normally exceed the equivalent of about 50 cc. N/10 acid per 100 cc. of stomach contents and will only rarely exceed twice that concentration even in cases of hyperchlorhydria.

Poisoning by Sulphuric Acid (H_2SO_4)

Source and Method of Occurrence. Sulphuric acid, or oil of vitriol, is more extensively used in the arts and manufactures than any other acid; hence cases of poisoning by it are common. Occasionally it is used for suicidal purposes, and accidents in consequence of its having been mistaken for some other liquid are numerous. It has often been used externally (vitriol-throwing, *vide* Vol. I. p. 505), with the object of injuring or disfiguring a person.

Toxicity and Fatal Dose. The effects of this acid, as of all corrosives and some irritants, depend not only upon the dose, but upon its concentration. The quantity required to prove fatal depends on many circumstances. If the stomach is full when the poison is swallowed, the main effect of the acid may be spent on the contents; and a larger quantity might then be taken than would suffice to destroy life if the stomach were empty.

The smallest quantity which is described as having proved fatal was in the following case:—

Half a teaspoonful of concentrated sulphuric acid was given to a child about a year old by mistake for castor oil. The usual symptoms came on, with great disturbance of breathing, and the child died in twenty-four hours.

The quantity here taken could not have exceeded **forty drops.**[1] It is, however, doubtful whether this small quantity would have proved fatal to an adult. The smallest fatal dose which Christison states he had found recorded is *one drachm;* it was taken by mistake by a stout young man, and killed him in seven days. Even when moderately diluted sulphuric acid may rapidly destroy life.

A man swallowed, on an empty stomach, six drachms of the strongest acid diluted with eighteen drachms of water. He suffered from the usual symptoms, and died in two hours and a half.[2]

Two-thirds of the cases prove fatal. The pharmacopœial dose is five to sixty minims of the dilute acid, which contains 10 per cent. of pure sulphuric acid.

Duration. When sulphuric acid is swallowed in a concentrated form, the symptoms produced come on either *immediately* or during the act of swallowing. In a case which came under the notice of Sir Thomas Stevenson, a girl swallowed about a fluid ounce of brown oil of vitriol in mistake for a magnesium mixture which she was in the habit of taking, and did not discover the mistake till severe symptoms had supervened. Generally, death occurs, in cases of acute poisoning by sulphuric acid, from **eighteen to twenty-four hours** after the acid is swallowed, but earlier than this if the stomach is perforated by the acid. In one instance, reported by Sinclair, a child about four years old died in four hours; the stomach was found perforated. When the poison acts upon the windpipe, death may be a still more speedy consequence from suffocation; and, owing to this, it appears to be more rapidly fatal to children than adults. Craigie mentioned a case in which three ounces of concentrated sulphuric acid destroyed life in three hours and a half.

[1] *Med. Gaz.*, vol. 29, p. 147.
[2] *Med. Times and Gaz.*, 1863, 1, p. 183.

Remer met with an instance in which death took place in *two hours*. A case is reported by Watson in which a woman swallowed two ounces of a strong acid. She died in *half an hour*, but it appears that a quarter of an hour before death she had made a deep wound in her throat, which gave rise to great bleeding. The stomach was found extensively perforated; but it is highly probable that the wound accelerated death in this case. The most rapid, uncomplicated, death was reported by Rapp. A man, *at* 50, swallowed three ounces and a half of concentrated sulphuric acid. He died in **three-quarters of an hour.**[1] On the other hand, there are numerous instances reported in which the poison proved fatal from secondary causes at periods varying from one week to several months. These secondary causes, such as contraction and occlusion of the gullet, may threaten, and even cause, death from starvation, and require operative procedures. When the passage of bougies to dilate the stricture of the œsophagus has failed to procure relief, gastrostomy has been successfully performed, and the patient fed through an artificial opening into the stomach.

Symptoms. There is violent burning pain (which begins as soon as the acid touches the mouth), extending down the throat and gullet to the stomach, and the pain is often so severe that the body is bent. There is an escape of gaseous and frothy matter, followed by retching and vomiting, the latter accompanied by the discharge of shreds of tough mucus and of a liquid of a dark coffee-ground colour, mixed with blood. The vomited matters may contain shreds of mucous membrane from the gullet and stomach, and even portions of the muscular structures of the former. These may form complete casts of some portion of the gullet or stomach. The mouth is excoriated, and the lining membrane and surface of the swollen tongue white, or resembling soaked parchment; in one instance the appearance of the mouth was as if it had been smeared with white paint. After a time the membrane acquires a grey or brownish colour; the mouth is filled with a thick viscid substance, consisting of saliva, mucus, and the corroded membrane; this renders speaking and swallowing difficult, and may cause death by asphyxia, especially if the epiglottis also becomes swollen. If the poison has been administered by a spoon, or the phial containing it has been passed to the back of the throat, the mouth may escape the chemical action of the acid. A medical witness must bear this circumstance in mind when he is required to examine an infant suspected to have been poisoned by sulphuric acid. Around the lips and on the neck may be found spots of a brown colour from the spilling of the acid and its action on the skin. There is great difficulty in breathing, owing to the swelling and excoriation of the throat and larynx, and the countenance has, from this cause, a bluish or livid appearance; the least motion of the abdominal muscles is attended with increase of pain. These symptoms, although characteristic of the action of a corrosive liquid, have been sometimes by carelessness mistaken for those of disease. The stomach is so irritable that whatever is swallowed is immediately ejected, and the vomiting is commonly violent and incessant. The matters *first* vomited generally contain the poison; they are acid, and if they fall on a limestone pavement there may be effervescence; if on coloured

[1] *Gaz. Med.*, December 28th, 1850.

articles of dress, the colour is sometimes altered to red or yellow, or it is entirely discharged, and the texture of the stuff destroyed; on the black cloth dress the spots produced by the concentrated acid are reddish brown, and remain moist for a considerable time. Attention to these circumstances may often lead to a suspicion of the real cause of the symptoms when the facts are concealed. After a time there is exhaustion, accompanied by great weakness and collapse; the pulse is quick, small, and feeble, the skin cold, mottled or dusky, and covered with a clammy sweat.

Vomiting may cease from the collapse for many hours before death actually takes place; there is generally great thirst, with obstinate constipation, and should any evacuations take place they are commonly either of a dark brown or leaden colour, in some instances almost black, arising from an admixture of altered blood. There are sometimes convulsive movements of the muscles, especially those of the face and lips. The countenance, if not livid from obstructed respiration, is pale, expressive of great anxiety and intense suffering. The intellectual faculties are quite clear, and in the greater number of cases of acute poisoning by this acid death takes place very suddenly in from eighteen to twenty-four hours after the poison has been taken:—

Walker met with a case in which a man, *æt.* 30, swallowed fifteen drachms and a half of sulphuric acid (sp. gr. 1·842), and died twenty-five hours afterwards. Half an hour after taking the poison he resembled a patient in the collapsed stage of cholera. The inside of the lips, as well as the tongue and throat, were swollen, and had the appearance of being smeared with thin arrowroot. He suffered severe pain, but did not vomit until *three-quarters of an hour* had elapsed; the vomiting appeared to be then excited by the liquid which had been given to him. The vomited matters were dark, bloody, and viscid. The patient was sensible up to the time of his death.

This case is remarkable in the fact that vomiting was not immediate; that there were no spots on the face; that the poison was swallowed in large quantity on an empty stomach; and there was free voluntary exertion, as twenty hours after he had taken the poison the man got out of bed and sat on a night-stool.

Treatment. Treatment is much simpler in theory than in practice, owing to the intense pain caused by swallowing. We must aim at speedy neutralisation of the acid; and for this purpose calcined magnesia is the most useful antidote. Bicarbonate of soda must be used if nothing else is handy, but it, like any other carbonate, such as chalk, has the obvious disadvantage that the carbonic acid liberated may tear a damaged stomach. However, something must be rapidly done, and these carbonates must be used if handy. Whitening used to clean silver may be easy to get, perhaps, and should be used. Lime-water may possibly be obtained. Plaster from the ceiling may be used, or whitewash from a wall, or egg-shells finely powdered. White of egg, soap and water, may be useful because easily obtainable. If none of these can be obtained, plain water must be given in considerable quantities. It will dilute the acid, and also render the vomiting less distressing. Milk or gruel will serve the purpose, or oatmeal stirred up in simple water.

On no account must attempts be made to use the stomach tube and emetics are assuredly not required.

Morphine should be given to relieve the pain, and belladonna or atropine may be of service in reducing gastric secretion and motility.

Intravenous saline perfusion with the addition of sodium bicarbonate or sodium lactate is necessary to replace fluids lost by vomiting and diarrhœa and to renew the alkali reserve.

After the first effects are over, demulcent drinks will assist, but this need not be further discussed. The case is then one of acute gastritis, to be treated on ordinary principles. The burns of the skin, if present, must be treated also on ordinary principles after all acid has been washed away or neutralised.

Post-mortem Appearances. The marked effects of this poison are usually observed in the stomach; but in rare instances they may be confined to the region of the throat and windpipe. In an inspection of the body the whole course of the alimentary canal from the mouth downwards should be examined, since in recent or acute cases it is in the throat and gullet that we generally obtain strong evidence of the action of a corrosive poison. The discovery of the usual marks of corrosion in these parts is always highly corroborative of the signs of poisoning found in the stomach. During the inspection the examiner must not omit to notice any spots on the skin produced by the spilling of the acid; these are commonly dark brown, and are situated about the mouth, lips, chin and neck. The appearances met with in the body vary according to whether death has taken place quickly or slowly. Supposing the case to have proved rapidly fatal, the membrane lining the mouth may be found white, softened, and corroded. The mucous membrane of the throat and gullet is commonly found corroded, having a brown-black or ash-grey colour, and dark-coloured blood is effused in patches beneath it. The corroded membrane of the gullet is occasionally disposed in longitudinal folds, portions of it being partly detached. In some cases an actual cast of the œsophagus has been expelled during life.[1] The stomach, if not perforated, is collapsed and contracted. On laying it open the contents are commonly found to be dark brown or black and of a tarry consistency, being formed very largely of mucus and altered blood. The contents may or may not be acid according to the time the patient has survived and the treatment which has been adopted. On removal the stomach may be seen to be traversed by black lines, or the whole of the mucous membrane may be corrugated, and stained black or dark brown. This blackness is not entirely removed by washing. On stretching the stomach inflammation may be found between the folds, indicated by a dark crimson colour. On forcibly removing the blackened membrane the red colour indicative of inflammation may be seen in the parts beneath. Both the dark colour and marks of inflammation are sometimes partial, being confined to isolated portions or patches of the mucous membrane. When the stomach is perforated, the coats are softened, and the edge of the aperture is commonly black and irregular. In removing the stomach the opening is liable to be made larger by the mere weight of the organ. Perforation does not always occur during life, but when this happens the surrounding parts are attacked by the poison. The spleen, the liver, and the coats of the aorta, have

[1] O. Wyss, "Arch. f. Heilk.," 1869, p. 184; Horneffer, "Diss. Greifswald," 1895.

been found blackened and corroded by the acid, which had escaped through the perforation. In rare cases the lining membrane of the aorta has been found strongly reddened. When a person has survived for eighteen or twenty hours, traces of corrosive and inflammatory action may be found in the small intestines. In one case the mucous membrane of the ileum was corroded. In fifty cases of sulphuric acid poisoning Lesser found corrosive changes in the small intestine in eighteen out of twenty-six cases that died acutely.[1] The interior of the windpipe, as well as of the bronchial tubes, has also presented marks of the local action of the acid. The acid has thus destroyed life without reaching the stomach. A remarkable instance in which the poison penetrated into and destroyed both lungs has been reported by Gull.[2] It is important for a medical witness to bear in mind that the mouth, throat, and gullet are not always found in the state above described. Ogle met with a case in which the membrane of the tongue was but slightly affected. The man had swallowed a large dose of the acid and had died in nine hours.[3] Many cases are recorded in which this poison has passed into the stomach, and yet the gullet has escaped.[4]

Recovery is rare even from the acute symptoms, and rarer still if we include recovery from the resultant gastric ulcer.

Analysis. This acid may be met with either concentrated or diluted ; and a medical jurist may have to examine it in three conditions : (1) in its simple state ; (2) when mixed with organic matters, as with liquid articles of food, or in the contents of the stomach ; (3) on solid organic substances, as where the acid has been thrown or spilt on articles of dress or clothing.

In the Simple State. The pure acid has a density of 1·84, but since it is hygroscopic, slight dilution caused by exposure to air may cause figures rather lower than this to be found. If *concentrated*, it possesses these properties : (1) Wood, sugar, or other organic matter plunged into it, is speedily carbonised or charred, either with or without the application of heat. (2) When boiled with wood, copper cuttings, or mercury, it evolves fumes of sulphurous acid ; this is recognised by the odour, as well as by the acid vapour first rendering blue, and then bleaching, starch-paper dipped in a solution of iodic acid. (3) When mixed with an equal bulk of water, great heat is evolved.

The Diluted Acid. For the acid in a diluted state but one test need be applied, that of a solution of a barium salt. Having ascertained by test-paper that the liquid is strongly acid, we dilute a portion of it and add a few drops of nitric acid and then a solution of the barium salt. If sulphuric acid is present, a dense white precipitate of sulphate of barium will fall down ; this is insoluble in all acids and alkalies. Alternatively, the acid may be neutralised with alkali and the solution evaporated to dryness, the residue heated on charcoal (whereby the sulphate is reduced to sulphide) and the product scraped on to a silver coin and moistened with dilute hydrochloric acid ; sulphuretted hydrogen will be liberated and form a dark brown stain of silver sulphide.

[1] *Virch. Arch.*, 83, 193.
[2] *Med. Gaz.*, vol. 45, p. 1102.
[3] *Med. Times and Gaz.*, 1860, 1, p. 408.
[4] Cp. 2 Casper, E. Tr., p. 83, and Hoffmann, Atlas, plates 33 and 34.

Dilute sulphuric acid does not carbonise organic substances which are immersed in it. The application of heat will only effect carbonisation when the water of dilution is in great part evaporated. Thus paper or linen, wetted with the dilute acid, becomes charred when dried and heated. This may serve as one method of identification in the absence of other, more precise, tests.

In Liquids containing Organic Matter. If sulphuric acid is mixed with such liquids as porter, coffee, or tea, the liquid is first cleared by filtration. Some liquids, such as vinegar, beer, and most wines, generally contain a soluble sulphate and have an acid reaction. Should the liquid be thick and viscid like gruel, it may be diluted with water, and then boiled with the addition of a little acetic acid. For the success of the barium test it is not necessary for the liquid to be absolutely clear, provided it is not so thick as to interfere mechanically with the subsidence of the precipitate.

A similar process may be applied to the examination of matters *vomited* and of the *contents of the stomach*, care being taken to separate the insoluble parts by filtration, before trying the test. The coats of the stomach should be cut up and boiled in distilled water for some time to extract the acid completely. The decoction, filtered and concentrated by evaporation, may then yield evidence of the presence of a free mineral acid (*vide* p. 290). If there be evidence of a free mineral acid, and the liquid yields a decided precipitate with solution of nitrate of barium, it should be concentrated by evaporation if necessary, and strong alcohol be added till the mass contains 75 per cent. by volume of absolute alcohol. The soluble sulphates are thus precipitated; and, after standing and filtration to remove these, the filtrate is neutralised with solution of potash, the alcohol distilled off, the solution evaporated to dryness. The residue, when taken up with water, may then be tested with nitrate of barium after acidification with nitric acid. One part of the barium sulphate precipitate corresponds to 0·42 part by weight of sulphuric acid.

Note, of course, that the soluble sulphates should be similarly estimated by precipitation and weighing of barium sulphate, for they may have been produced *in situ* by neutralisation of the free acid and the question of whether sulphuric acid has been administered may have to be decided on the basis of the total amount of sulphate found (together with the symptoms and post-mortem findings). Only small amounts of sulphate are normally present. Some sulphate, actually combined with protein, will not be extracted by this method.

When the acid is mixed with milk, decomposed blood, and mucus, or other substances which render it thick and viscid, it may be readily separated by **dialysis,** a process which is applicable to the other acid poisons, such as nitric, hydrochloric, and oxalic. A portion of the acid viscid liquid should be placed in a test-tube, about five inches long and one inch in diameter, open at both ends, the mouth being securely covered with a layer of thin bladder or parchment paper. The tube is then immersed, mouth downwards, in a vessel containing distilled water. After some hours the acid will pass through the membrane, and may be detected in the water. The dialysis will be quicker if, instead of a glass tube with only the mouth as a dialysing surface, a sac of

collodion or cellophane is used. This process may be employed as a trial test of the contents of the stomach when they have a strongly acid reaction. In thus testing for sulphuric acid it must be remembered that a sulphate, such as Epsom salts, may be present in the liquid, and an acid such as vinegar or lemon-juice may give the acid reaction. To remove any fallacy on this ground, a portion of the liquid tested should be evaporated, and treated with alcohol as already described. Complete differentiation of sulphuric acid and organic salts from soluble sulphate and organic acid may not be possible, but can sometimes be done by quantitative analysis and sometimes more simply by the alcohol treatment already described.

It is an important fact that the contents of a stomach in a case of poisoning by sulphuric acid are sometimes entirely free from any traces of this poison, even when it has been swallowed in large quantity. This is the result of vomiting and treatment. In many cases of poisoning by sulphuric acid, therefore, a medical witness must be prepared to find that chemical analysis will furnish only negative results. This, however, is not inconsistent with death having taken place from the poison.[1]

If the stomach should be perforated, the contents will be found in the abdomen, or perhaps in the lower part of the cavity of the pelvis. They may then be collected, boiled with distilled water, and the solution examined for the acid by the process already described. If the contents of the stomach are highly putrefied, the sulphuric acid may be found combined with ammonia.

On Solid Organic Substances. It sometimes happens in cases of poisoning by sulphuric acid that it is spilled upon articles of clothing, such as cloth or linen, and here a medical jurist may succeed in detecting it when every other source of chemical evidence fails. Again, sulphuric acid is often used for the purpose of seriously injuring a person by throwing it on him (*vide* Vol. I, p. 505, "Vitriol-throwing"). The process of analysis is simple. The spot, unless it has been washed gives a blue colour with moist congo-red paper. The stained fabric should be digested in rectified spirits at a gentle heat, whereby a brownish-coloured liquid may be obtained on filtration. If sulphuric acid is present, the liquid will have an acid reaction, and produce the usual effects with the barium test after evaporation and dilution with water. Old stains are known by the complete destruction of the organic fibre, fresh stains by their dampness. The acid remains fixed in the stuff. Sulphuric acid has been thus detected in clothing after the lapse of twenty-seven years. The detection of spots of this acid on articles of dress has in some cases served to supply the place of direct evidence from a chemical analysis of the stomach; and in other instances it has aided justice in fixing on an accused person the act of administration.

Sulphate of Indigo

Source and Method of Occurrence. This compound is a commercial product, and consists essentially of a solution of indigo in strong sulphuric acid. Several cases of accidental poisoning by it have occurred.

[1] Cp. 2 Casper, E. Tr., p. 77 and p. 81

As indigo is one of the substances directed to be mixed with arsenic when this poison is sold in small quantities, the detection of this colouring principle in the mouth and vomited matters will not necessarily show that it has been taken in the form of sulphate.

Toxicity and Fatal Dose ; Duration ; Symptoms ; Treatment. These are similar to those which have been already described for sulphuric acid, which is the toxic agent. This kind of poisoning may be suspected when, together with these symptoms, the membrane of the mouth has a blue or blue-black colour. The vomited matters, as well as the fæces, are at first of a deep blue-black tint, afterwards green ; and it was observed in two instances that the urine voided by the patients had a blue tinge.

Analysis. The solution is of a dark blue colour, and strongly acid. Sulphuric acid is detected in it by the methods above described. The blue colour is discharged by chlorine, or when a portion previously diluted is boiled with nitric acid.

Poisoning by Nitric Acid (HNO_3)

Source and Method of Occurrence. Nitric acid is popularly known under the name of aquafortis, or red spirit of nitre. According to Tartra, it seems to have been first used as a poison about the middle of the fifteenth century. Although it is perhaps as much used in the arts as oil of vitriol, cases of poisoning by it are not very common.

Toxicity and Fatal Dose. These depend more upon the concentration than upon the quantity of the acid taken. The *smallest* quantity reported to have destroyed life is about *two drachms*, administered to a boy, aged thirteen, who died in about thirty-six hours. But less than this quantity, even one drachm, might doubtless suffice to cause death, for the fatal result depends on the extent of the damage to the air passages, gullet, and stomach. It is difficult to state the largest dose of concentrated acid from the effects of which a person has recovered, since in most of the cases of recovery reported the quantity of the poison taken was unknown. A case of recovery from about half an ounce of the strong acid mixed with the diluted acid is reported.[1] The patient was a man *æt.* 21. He had the usual symptoms, with the exception that there was no yellowing of the teeth, nor corrosion of the mouth. The vomited matters were bloody and of a dark colour. He suffered from stricture of the gullet, and this remained when he left the hospital about fifteen weeks after admission.

Duration. The symptoms of acute poisoning appear while swallowing the fluid if it be at all concentrated, but when the dilute acid is taken the time of onset of symptoms and their severity will depend largely on the condition of the stomach as regards its contents. Sobernheim relates a case of poisoning by nitric acid which proved fatal in **one hour and three-quarters.**[2] This we believe to be the most rapidly fatal instance on record in an adult. The usual well-marked effects were found in the gullet, stomach, and small intestines. In infants, however, life may be destroyed by this poison in a few minutes should it happen to affect the

[1] *Lancet*, 1870, 1, p. 549.
[2] *Op. cit.*, 402.

larynx. Death commonly takes place in from eighteen to twenty-four hours, and the intellectual faculties commonly remain clear until the last. Sometimes death is preceded by a kind of stupor from which the patient is easily aroused. In one case the patient was insensible, but she ultimately recovered. Death may take place months or years afterwards from the effects of the ulcers or strictures caused by the acid.

Symptoms. When nitric acid is taken in a concentrated state, the symptoms, on the whole, bear a close resemblance to those produced by sulphuric acid. They come on *immediately*, and the swallowing of the acid is accompanied by intense burning pain in the throat and gullet extending downwards to the stomach. There are gaseous eructations from the chemical action of the poison, swelling of the abdomen, violent vomiting of liquid or solid matters, mixed with altered blood of a dark brown colour, and shreds of yellowish-coloured mucus, having a strongly acid reaction. The abdomen is generally exquisitely tender, but in one well-marked case of poisoning by the acid the pain was chiefly confined to the throat ; probably the poison had not reached the stomach. The mucous membrane of the mouth is commonly soft and white, after a time becoming yellow, or even brown : the teeth are also white or yellow, and the enamel is partially destroyed by the chemical action of the acid. There is great difficulty in speaking and swallowing, the mouth being filled with viscid mucus : the power of swallowing is sometimes lost. On opening the mouth the tongue may be found swollen, and of a citron colour ; the tonsils are also swollen and enlarged. The difficulty of breathing is occasionally such as to render tracheotomy necessary, especially in young persons. As the symptoms progress collapse supervenes. The administration of remedies, even the swallowing of the smallest quantity of liquid, increases the severity of the pain, occasions vomiting, and gives rise to a feeling of laceration or corrosion. There is obstinate constipation. Death may be occasioned by the action of this acid on the larynx, as in the case of sulphuric acid. Should the patient survive the first effects of the poison, the mucous membrane of the throat and gullet may be ejected, either in regular masses or in the form of a complete cylinder. The stomach may be ulcerated and this may lead ultimately to death.

There is one very important particular in which nitric and hydrochloric acids differ from sulphuric, *viz.*, in their volatility, so that at the ordinary temperatures of the air they give off fumes which will cause serious and even fatal injury to the mucous membrane of the air-passages. In these cases the symptoms are those of intense suffocative bronchitis, frequently with lung œdema. They often appear several hours after inhalation of the poison. The fumes associated with nitric acid (probably of the acid itself mixed with oxides of nitrogen) are very deadly. Such fumes are evolved during the manufacture of explosives such as gun-cotton and if respired, though they may have no obvious immediate effect, are liable to cause severe bronchitis and, ultimately, death. The victims of poisoning by exposure to nitrous fumes are often recognisable by the yellow colour of hair, eyebrows, and even of the exposed parts of the skin. The teeth may show signs of acid necrosis.

Treatment. The same as for sulphuric acid.

Post-mortem Appearances. If death occurs quickly from the effects of the liquid acid, the skin of the mouth and lips is deeply stained yellow-orange-brown and, as after burning or scalding, is easily detached (splashing or spilling may have produced similar yellow spots or patches on the hands, neck, etc.). Yellow spots produced by the spilling of the acid may be found about the hands and neck. A yellow frothy liquid escapes from the nose and mouth; and the abdomen is often much distended. The membrane lining the mouth is sometimes white, sometimes citron coloured; the teeth are white, but present a yellowish colour about the coronæ. The pharynx and larynx are much inflamed; and the latter is sometimes œdematous. The lining membrane of the gullet is softened, and of a yellow or brown colour, easily detached, often in long folds. The windpipe is inflamed, the lungs are congested and œdematous, and there may be patches of necrosed mucous membrane in the bronchial tubes. The most strongly marked changes are, however, seen in the stomach. When not perforated, this organ may be found distended with gas, its mucous membrane inflamed, and covered by patches of a yellow, brown, or green colour, or it may be even black. This green colour is due to the action of the acid on the colouring matter of the bile; but it must be remembered that a morbid state of the bile itself may give a similar appearance to the mucous membrane in many cases of death from natural disease. The stomach coats may be so softened as to rupture with the slightest pressure, and there may be commencing peritonitis, although no perforation has taken place. In the duodenum similar changes are found; but in some cases the small intestines have presented no other appearance than that of slight redness. It might be supposed that the stomach would be in general perforated by this corrosive liquid; but perforation has not often been observed. The question of perforation is entirely one of quantity and strength (concentration) of the acid, conditions obviously influenced by food in the stomach. In those cases in which death takes place at a remote period ulcers and cicatrices will be found, only to be distinguished from idiopathic ones by their irregularity of shape and distribution, unless there is a history of previous poisoning.

Analysis. *In the simple state.* This acid may be met with either concentrated or dilute. The *concentrated acid* varies in colour from a deep orange red to a light straw yellow. It may be recognised, 1. By evolving yellow-brown acid fumes when exposed to the air or when heated. 2. By its staining nitrogenous organic matter, such as wool, yellow or brown, the colour being heightened and turned to an orange-red tint by contact with caustic alkalies. 3. When mixed with a few copper cuttings, it is rapidly decomposed, deep red acid vapours are given off, and a bluish-green solution of nitrate of copper is formed. Tin or mercury may be substituted for copper in this experiment. *In the dilute state.* This acid is not precipitated, like sulphuric, by any common reagent, since all its salts are soluble in water. 1. The liquid has a highly acid reaction, and on boiling it with some copper turnings, red nitrous fumes are given off, unless the proportion of water is very great. At the same time the liquid acquires a blue colour. 2. A streak made on white paper with the diluted acid does not carbonise the paper when heated; but a faint yellow stain is left. 3. The liquid is

not precipitated by salts of barium or of silver. The two last experiments give merely negative results: they serve to show that sulphuric and hydrochloric acids are absent.

In order to detect nitric acid, the liquid should be first tested for the presence of a free mineral acid in the way described (p. 290); then tested for the presence of nitrates as below.

In liquids containing organic matter. Nitric acid may be administered in such liquids as tea, vinegar, or beer. In this case, besides the acid reaction, there will be a peculiar smell produced by the strong acid, when mixed with substances of an organic nature. The application of the usual tests may be here counteracted: thus, unless the quantity of nitric acid in the liquid is rather large, the orange-red nitrous fumes are not evolved on boiling it with copper cuttings. The action on gold-leaf will enable the chemist to detect nitric acid in coffee, tea, and similar organic liquids, even when the proportion of acid is small. Boil a fragment of gold-leaf in pure hydrochloric acid, and add while boiling a few drops of the suspected organic liquid to the mixture. If the acid is present the gold will be dissolved. When the acid liquid is thick and turbid, a portion of it may be submitted to dialysis. Vomited matters, as well as the contents and coats of the stomach (cut up), should be boiled in water, and filtered. If not cleared by filtration, they may be submitted to dialysis, and the acid water obtained neutralised by potash, and concentrated. If by filtration we succeed in procuring a clear acid liquid, the colour is of no importance. The liquid should be carefully neutralised with a solution of pure potash, and concentrated by evaporation. Drops of this may be placed on a slide, and the crystals microscopically examined and compared with those of nitre. Paper dipped into the concentrated neutralised liquid, and dried, burns like touch-paper. The crystals, on heating, evolve oxygen and leave potassium nitrite which, when warmed with concentrated sulphuric acid, evolves red acid fumes (mainly nitrogen peroxide).

The crystals obtained by evaporating the neutralised liquid are generally coloured with organic matter, but they fuse into a white mass when carefully heated in a platinum capsule. (Overheating, however, converts nitrate to nitrite.) The pure nitre thus obtained may be tested as above described. The organic matter in the crystals does not interfere with the results of the copper and gold tests.

When either the nitric acid, or the nitrate into which it has been converted, is mixed with common salt, the copper test cannot be employed. The gold test will in such a case furnish the best evidence. Hydrochloric acid with a small portion of gold-leaf may be added to the dried residue, and the mixture boiled. If nitric acid or a nitrate is present, even in minute proportion, some portion of the gold will be dissolved, a fact demonstrable by the addition of stannous chloride.

Nitric acid may be detected in *stains on clothing*, if recent, by simply boiling the stained cloth in water. An acid liquid will be obtained, unless the stains are of old date or the stuff has been washed. This liquid, when concentrated, may be dealt with in the manner already described. The stains from this acid on black and blue cloth are of yellow or brownish-yellow colour. When long exposed they become dry, but the cloth is easily torn. A simple method of detecting the acid

is to boil at once a piece of the stained cloth with a fragment of gold-leaf and hydrochloric acid. If nitric acid or a nitrate is present in the stain, a portion of the gold will be dissolved.

In most cases of poisoning by nitric acid the amount of acid involved is so large that the relatively crude tests so far described are adequate. The three following tests are very delicate and may be used as confirmatory tests or for dealing with very small quantities of material. It must, however, be remembered that traces of nitrate or nitrite may be present in suspected material from causes other than poisoning by nitric acid.

1. If to a nitrate a few drops of strong sulphuric acid are added, and then a crystal of Brucine stirred into the mixture, a bright red colour is produced. This test is so delicate that even a considerable amount of foreign colouring material does not obscure it, though, of course, the less colour from other sources the better the blood-red colour of the Brucine reaction is seen.

2. If to a test-tube containing a colourless solution of a nitrate a few crystals of ferrous sulphate be added and the test-tube shaken for a moment or two and then some strong sulphuric acid be added in such a manner that the sulphuric acid fills the bottom of the test-tube, then at the junction of the sulphuric acid and the liquid to be tested a dark purplish-brown, almost black, ring will develop; moreover, around those crystals of the ferrous sulphate which have not completely dissolved a similar brown or black ring will develop. This test, though very delicate in colourless solutions, is useless in deeply coloured ones. (Nitrites give this reaction.)

3. Diphenylamine test.—Diphenylamine $\left(^{C_6H_5N}<^{H}_{C_6H_5}\right)$ freshly dissolved in concentrated sulphuric acid (0·1 grm. in 10 c.c.) gives with nitric acid or its salts a blue zone where the fluids mix. This is a very sensitive test, 0·5 mgr. of HNO_3 in 1 litre of blood giving a definite reaction. (Nitrites also give this reaction.)

Cases. A chemist was pouring a mixture of nitric and sulphuric acids from a carboy containing about sixty pounds, when by some accident the vessel was broken. For a few minutes he inhaled the fumes of the mixed acids, but it does not appear that any of the liquid fell over him. Three hours after the accident he was sitting up and appeared to be in moderately good health. He was then seen by a medical man, and complained merely of some cuts about his hands. He coughed violently. In three hours more there was difficulty of breathing, with increase of the cough. There was a sense of tightness at the lower part of the throat, and the pulse was hard. At times he said he could scarcely breathe. He died eleven hours after the accident. On inspection, there was congestion of the windpipe and bronchial tubes, with effusion of blood in the latter. The heart was flaccid, and contained but little blood; and the lining membrane of the heart and aorta was inflamed. The blood had a slightly acid reaction.

A similar accident occurred to Mr. Stewart and one of the janitors of an educational institution in Edinburgh, in 1863. A jar of nitric acid, which he was carrying, fell on the floor and was broken. He and the janitor, instead of withdrawing from the spot, wiped the floor, and attempted to save some of the acid. They thus inhaled the fumes which were immediately diffused. Stewart returned home unconscious of the mischief which had been done. After an hour or two, difficulty of breathing came on, and in spite of every effort to save his life, he died in ten hours after the accident. The janitor suffered from similar symptoms, and died the day following.[1]

Taylor recorded that, on one occasion when he was preparing gun-cotton, he accidentally inhaled the vapour and suffered from severe constriction of the throat, tightness in the chest, and cough for more than a week. The fumes from batteries worked with nitric acid are often productive of serious results, and Sir Thomas Stevenson has met with serious cases resulting from their inhalation.

In 1888 a man was convicted of the murder of a woman by pouring nitric acid down her throat whilst in bed.[2] In reality a mixture of sulphuric and nitric acids, containing more sulphuric than nitric, was employed. Such a mixture does not char cellulose (wood and cotton) like sulphuric acid, but converts it

[1] *Chem. News*, 1863, p. 132.
[2] *R. v. Lipski*, C.C.C., July 1888.

into nitro-cellulose, which substance was found by Sir Thomas Stevenson in the stains on the deceased woman's linen, and also in wood cut from the floor of the room in which the murder was committed.

A serious fire, caused by the bursting of a carboy filled with nitric acid, broke out on the premises of a well-known chemical factory. The conflagration involved the whole building where the stock of this chemical was kept, and a high wind drove the smoke and fumes towards the firemen, who were immediately seized with a violent cough, dyspnœa, nausea, and headache, rendering it necessary for them to be relieved every few minutes. The men were able to continue on duty and to walk home when the fire was over, but after several hours thirteen of them developed symptoms of poisoning, eleven were taken to the hospital, whilst two died before they could be admitted. The symptoms differed much in their intensity, but all the patients suffered from dyspnœa, vomiting, cyanosis of the skin, sopor, and clonic convulsions. The heart's action was at first accelerated, but within a few hours became very slow, making only from forty-five to fifty-four beats per minute; sometimes the pulse was irregular. All the patients complained of oppression in the chest and great pain near the sternum. This was followed by severe bronchitis, the expectoration being sometimes of a yellow colour and sometimes tinged with blood. Some of the patients had a difficulty in swallowing, due to inflammation of the fauces. Albumen was found in the urine of three of them for periods varying from three to eight days. Seven recovered completely within ten days, but the remaining four were very weak and off duty for about a fortnight longer.[1]

The two following cases show the danger of the fumes from nitric acid:—

An inquiry was held at Aston on May 5th relative to the death of a man described as an annealer and dipper. His duty appears to have been that of dipping copper cups in dilute nitric acid after they had been annealed and pickled. The medical evidence pointed to acute congestion of the lungs, a condition consistent with the statement that the deceased had inhaled acid fumes. The jury returned a verdict in accordance with this and added that they did not attach any blame to any one, but recommended the company to supply chemically charged respirators for the men employed in work similar to that which the deceased did and to have printed notices exhibited to that effect. The product of the action of dilute nitric acid on copper is, of course, nitric oxide, which, however, in contact with air forms nitric peroxide. This dark red fume meeting the moisture of the respiratory passages would split up into nitrous and nitric acids, which are distinctly injurious to the lung tissues.[2]

On September 19th a man, 32 years of age, employed by Messrs. Curtiss and Harvey in the manufacture of nitro-glycerine, was admitted into St. Bartholomew's Hospital, Rochester, and died an hour later. He was too distressed to give any account of what had caused his illness. On admission he was cyanosed, with laboured and shallow breathing. His pulse rate was 120, his respirations were 42, and his temperature was 98° F. He was put to bed surrounded with hot-water bottles, strychnine was given hypodermically, and oxygen was administered. On two occasions he coughed up a small amount of frothy bloodstained fluid. His heart continued to beat for a short while after respiration ceased. At the necropsy the pleural cavities were found to contain a good deal of fluid: the lungs were very congested and full of frothy fluid; the blood was very dark in colour and had not coagulated. Nitro-glycerine is made by treating glycerine with sulphuric and nitric acids. The waste acid left at the end of the process is said to contain 70 per cent. of H_2SO_4, 10 per cent. of HNO_3, and 20 per cent. of water. When all nitro-glycerine had been skimmed off the acid liquid was made to flow along an open drain through which water had been running. The man neglected to shut off the water beforehand, and when the acid liquid came in contact with the water it boiled over. He, to conceal his omission, wiped up the mess and inhaled fumes

[1] *Lancet* 2: 226, 1897.
[2] *Lancet*, 1898, 1, p. 1341.

of nitric oxide and nitric peroxide, into which the nitric acid is said to be broken up by the heat. This, it was stated, happened at 8 a.m., and the man continued at work until 12 noon, when illness compelled him to stop.[1]

Poisoning by Hydrochloric Acid (HCl)

Source and Method of Occurrence. Popularly known as spirits of salt, hydrochloric acid is an important commercial commodity. It causes a certain number of deaths from accident, and is used by a number of suicides.

Toxicity and Fatal Dose. The smallest quantity of hydrochloric acid which has as yet been known to prove fatal was a **teaspoonful** in a girl fifteen years of age[2] Cases have been recorded of death in adults from taking half an ounce of the commercial acid, but on the other hand recovery has taken place after the ingestion of an ounce.[3] The pharmacopœial dose is five to sixty minims of the dilute acid, which contains 10 per cent. of HCl.

Duration—Symptoms—Treatment. These are similar to those of nitric acid, including the symptoms due to its volatility.

Post-mortem Appearances. These resemble those of nitric acid, but excluding the deep yellow-brown staining caused by the characteristic action of nitric acid on protein. The skin and tissues may be whitened, or, if the acid is in concentrated state, blackened. According to experience, too, it has less tendency than the other two strong mineral acids to perforate the stomach.

Analysis. The liquid known as hydrochloric acid is a solution of hydrochloric acid gas (HCl) in water, and may contain as much as 35 per cent. of the dissolved gas. In *a concentrated* state, hydrochloric acid evolves copious fumes, and the fumes become much denser if a glass stopper moistened with a strong solution of ammonia is brought near. The pure acid is nearly colourless; the commercial acid is of lemon-yellow colour, and frequently contains iron, arsenic, common salt, and other impurities. Some commercial samples are contaminated with benzol, and smell strongly of it. It reddens litmus paper strongly and gives the general reactions of a mineral acid. When boiled with a small quantity of black oxide of manganese, chlorine is evolved. It does not dissolve gold-leaf until a few drops of nitric acid have been added to it, and the mixture is heated. In the *diluted* state, these properties are lost. It gives a dense white precipitate when a solution of nitrate of silver is added to it. This precipitate is insoluble in nitric acid, but soluble in ammonia; it acquires a purple and black colour if exposed to light, and when heated it melts without decomposition, forming a yellowish-coloured substance on cooling. If the acid is contained in organic liquids in moderate quantity, it can be separated by distillation or dialysis. In this case any fixed chlorides present are left in the retort. Chlorides, which may be present normally, react with sulphuric acid to give hydrochloric acid, so that before relying on the results of distillation, therefore, it is necessary to show, not only that hydrochloric acid is distilled off, but also that the original material contained no sulphuric acid.

[1] *Ibid.*, 1902, 2, p. 1163.
[2] *B.M.J.*, March, 1871.
[3] *Lancet*, 1850.

Hydrochloric acid, in small quantity, and alkali chlorides, are natural constituents of the fluids of the stomach and bowels. The presence of local chemical changes in the throat and stomach would show whether the acid had been taken as a poison. If the acid is found only in minute quantity, no inference of poisoning can be drawn unless there are distinct marks of its chemical action upon the throat and stomach. It darkens the blood like sulphuric acid, although it has not the same carbonising action on organic matter. The *stains* produced by this acid on black cloth are generally of a reddish colour. As the acid is volatile, it may possibly disappear from the stuff. If the stain is recent, the acid may be separated by boiling the stuff in water and applying the silver test, or by boiling a portion of the stained cloth with gold-leaf and nitric acid. An unstained portion of cloth should be similarly tested for the sake of comparison, as neutral chlorides give a similar precipitate with nitrate of silver.

Poisoning by Hydrofluoric Acid (HF)

Source and Method of Occurrence. The acid is manufactured for the purpose of etching on glass.

Toxicity and Fatal Dose. The acid is a violent poison, both in itself and also because the commercial acid contains sulphuric acid. As usually supplied to glass engravers the acid contains about 40 per cent. of HF.

The dose in the case quoted below was uncertain, "a tablespoonful" or "a half-quartern" being the estimates of witnesses.

Duration. Death takes place within an hour or two.

Symptoms. Vomiting and collapse are the principal symptoms.

Inhalation of the gas produces ulceration of the conjunctiva and mucous membrane of the nose and mouth. Inflammation of the larynx and bronchitis have also been reported.

Treatment. Alkalies, milk and demulcent drinks should be given and general measures taken to counteract shock.

Post-mortem Appearances. *Blood*—Very tarry, but without clots; *lips*—Very charred; *tongue*—Sides denuded of papillæ, dorsum brownish, but not much burnt, back part of the epiglottis and fauces a deep brown colour, congested and ecchymosed; *pharynx*—Purplish slate colour, ecchymosed and congested, tissues round the rima glottidis were reddened and ecchymosed; *œsophagus*—Much congested, the whole of a slate colour, with deep red patches; *stomach*—Cardiac portion markedly ecchymosed, slight ecchymosis towards the pyloric orifice, no perforation and no denudation of mucous membrane of stomach; *intestines*—Nothing abnormal could be detected; *lungs*—Both very much congested and almost black in colour.

Case. A sign-writer and glass embosser had completed some work and retired to a public-house for some refreshment, having with him his tools and indiarubber bottle of hydrofluoric acid, used in his employment. He poured out a tablespoonful or "a half-quartern" of the acid into a glass and diluted it with water, remarking to the barmaid, "This is as harmless as the liquor you sell," and put it to his lips. Shortly after he was seen to be looking very white and clammy, and assistance was obtained. He was able to speak, and said that he had taken it before, but now had taken too strong a dose, and asked for milk to kill the acid.

This was given him, but without effect, and after some vomiting, he was removed to hospital, where he died about an hour afterwards, previously stating that he had taken it "for foolery."

His wife, in evidence, stated that she had heard her husband say that one drop of the acid was good when taken medicinally, but he knew it was a deadly poison; he had never threatened suicide.[1]

Stevenson examined the acid used in this case, and found that it contained 9·2 per cent. only of HF, whereas the commercial acid as sent out to glass engravers is usually at least four times this strength. The quantity of the acid taken in this case cannot be stated with any certainty; it may have been as much as two fluid ounces or as little as half an ounce of the solution (9·2 per cent.).

A case is reported in which half an ounce was taken, and death occurred in two hours.[2]

Huppert[3] records a case of poisoning in a worker in a mineral water factory who drank etching liquid. He died in an hour without special clinical phenomena being noted. The mucous membrane of the pharynx, œsophagus, stomach, duodenum and jejunum was of a grey colour, and swollen.

Two fatal cases of poisoning by silico-fluoride commonly sold as insect powder are reported by Lührig.[4] In one of these half a spoonful of the salt was taken by mistake. Fluorine was found in various organs including the kidneys and liver.

Poisoning by Oxalic Acid (COOH)

Source and Method of Occurrence. Oxalic acid is commonly used for cleaning brass, etc., also in the bleaching of straw. It is also a constituent, though in small amount, of sorrel leaves and some species of Rumex. It occurs, too, in rhubarb, and recent events seem to show that the green leaf contains quantities that are certainly noxious and may be fatal. Accidents to children from eating sorrel are occasionally reported (*vide infra*, "Vegetable Poisons").

Toxicity and Fatal Dose. The smallest quantity of this poison which has been known to destroy life is **sixty grains**. A boy, *æt.* 16, took the acid in a solid form, and in about an hour was found insensible, pulseless, and his jaws spasmodically closed. He had vomited some bloody matter: his tongue and lips were unusually pale, but there was no excoriation. He died in eight hours.[5] In one case *three drachms* destroyed life in an hour. Two cases occurred at Guy's Hospital, in each of which half an ounce of oxalic acid had been swallowed. Active treatment was adopted, and both patients recovered. When the dose is upwards of half an ounce, death is commonly the result; but a case has occurred in which a man recovered after having taken an ounce of the acid. The acid was in this instance taken by mistake for Epsom salts. Ellis met with a case in a woman, *æt.* 50, who swallowed an ounce of the acid in beer. In half an hour she was found complaining of a burning pain in the stomach, and rolling about. Chalk and water were freely given, and she recovered.[6]

[1] *B.M.J.*, October 21st, 1899.
[2] *Lancet*, February 8th, 1893.
[3] *Deut. Zeits., f. d. Gesamte Gerichtlich Med.*, 1926, 8, 424.
[4] *Chem. Zeit.*, 1925, 49, 805.
[5] *Lancet*, 1855, 2, p. 521.
[6] *Lancet*, 1864, 2, p. 265.

Duration. In oxalic acid we have a typical illustration of a poison that acts in two distinct ways—first of all by its immediate corrosive action, and secondly, by its effects after absorption. This double action explains the fact that similar quantities of this poison do not always destroy life within the same period of time. In two cases, in each of which about two ounces of the acid were taken, one man died in twenty minutes—the other in three-quarters of an hour. Christison mentions an instance in which an ounce killed a girl in thirty minutes, and another in which the same quantity destroyed life in *ten minutes*, but in a third case death did not occur until the fifth day. Ogilvy reported a case of poisoning by oxalic acid, in which it is probable that death took place within *three minutes* after the poison had been swallowed. The sister of the deceased had been absent from the room about that period, and on her return found her dying; the quantity of poison taken could not be determined. "Almost immediately" is a very common statement of the rapidity of death. Sir Thomas Stevenson had met with cases of death in *fifteen* and *twenty minutes* respectively. When the dose of oxalic acid is half an ounce and upwards, death commonly takes place within an hour. There are, it must be admitted, numerous exceptions to this rapidity of action. Christison reported two cases which did not prove fatal for thirteen hours; and in an instance that occurred to Fraser, in which half an ounce was taken, the individual died in a state of exhaustion from the secondary effects thirteen days after taking the poison. The great differences observed must obviously depend in part upon the condition of the stomach as regards its readiness for absorption. Oxalic acid is freely soluble in water, and if swallowed in solution on a moderately empty stomach, in which absorption is freely proceeding, it is easy to conceive that death might very rapidly take place from the effect of the absorbed acid on the heart. It is probable that its action after absorption is due to the fact that it precipitates calcium from the body fluids.

Symptoms. If this poison is taken in a large dose, *e.g.*, from half an ounce to an ounce of the crystals dissolved in water, a hot burning acid taste is experienced during the act of swallowing the poison. This is accompanied by a similar sensation extending through the gullet to the stomach. There is sometimes a sense of constriction or suffocation: the countenance is livid, and the surface of the skin soon becomes cold and clammy. Vomiting occurs either immediately or within a few minutes. Should the poison be diluted, there is merely a sensation of extreme soreness, and vomiting may not occur until after a quarter of an hour or twenty minutes. In some cases there has been little or no vomiting, while in others this symptom has been incessant until death. In a case in which an ounce of the acid was swallowed, the vomiting and pain in the stomach continued until the fifth day, when the man died suddenly.[1] In a case in which the poison was much diluted, vomiting did not occur for seven hours. The vomited matters are highly acid, and have a greenish-brown or almost black colour; they consist chiefly of mucus and altered blood. In one reported instance they were colourless.[2] In another case fluid blood of a bright arterial colour was

[1] *Lancet*, 1860, 2, p. 509.
[2] *Med. Gaz.*, vol. 27, p. 792.

vomited after some hours.[1] There is great pain and tenderness in the abdomen, with a burning sensation in the stomach. There are cold clammy perspirations and convulsions. In a case in which about two ounces of the poison had been swallowed there was no pain. Violent vomiting and collapse were the chief symptoms. There is in general an entire prostration of strength, so that if the person is in the erect position he falls; there is likewise unconsciousness of surrounding objects, and a kind of stupor, from which, however, the patient may be roused without difficulty. Owing to the severity of the pain, the legs are sometimes drawn up towards the abdomen. The pulse is very feeble, small, irregular, and scarcely perceptible. The blood-pressure falls. There is a sensation of tingling or numbness in the extremities, and shortly before death the respiration is spasmodic. The inspirations are deep, and a long interval elapses between them. Such are the symptoms commonly observed in a rapidly fatal or acute case. In the majority of fatal cases death takes place within an hour.

Should the patient survive the first effects of the poison, the following symptoms may appear: there is soreness in the mouth, constriction and burning pain in the throat, pain in swallowing, tenderness in the abdomen, and irritability of the stomach, so that there is frequent vomiting, accompanied by purging. The tongue is swollen, and there is great thirst. The patient may slowly recover from these symptoms. In one case the patient lost her voice for eight days. A man swallowed a quarter of an ounce of the acid and suffered from the usual symptoms in a severe form. In about nine hours his voice, although naturally deep, had become low and feeble. The weakness of voice remained for more than a month, and its natural strength had not returned even after the lapse of nine weeks. During the first month there was numbness with tingling of the legs.[2] Stevenson saw a similar case, in which a man almost lost his voice for some time. The occurrence of the sensation of numbness, and its persistence for so long a period after recovery from the symptoms of irritation, point to the effect of the poison on the nervous system. Spasmodic twitchings of the muscles of the face and extremities have also been observed in some instances,[3] and even convulsions of a general type. Since oxalic acid is a powerful kidney irritant, symptoms of acute nephritis may be expected if the patient survives sufficiently long. The urine passed subsequent to the poisoning usually contains albumen and blood, and the sediment contains octahedral crystals of calcium oxalate. The secretion of urine commonly decreases, and there may even be anuria.

The mortality in 242 reported cases was 54·5 per cent.[4]

Treatment. Calcium oxalate is the most insoluble oxalate, and hence chalk or calcined magnesia should be given at once. Saccharated solution of lime is very useful if at hand, but no time should be lost in giving some form of lime to neutralise the acid. Sodium or potassium salts must not be given, for their oxalates are very soluble and poisonous, hence the administration of these alkalies may defeat its own purpose.

[1] *Prov. Jour.*, June 25th, 1851, p. 344.
[2] *Med. Times*, 1850, 2, p. 293.
[3] *Lancet*, 1851, 1, p. 329.
[4] Brown and Gettler, *Jour. Exp. Biol. and Med.*, February 1922.

water again, which might dilute the acid, is likely only to promote a more rapid absorption, particularly if warm water is used.

Again, as regards emptying the stomach, it is better to avoid the use of the stomach tube owing to the possibility of erosion having taken place; but if vomiting has not taken place, the stomach should be washed out by the stomach tube, great care being exercised in passing it. Emetics are not likely to be of service, and a hypodermic injection of apomorphine would be risky on account of its depressing effect.

Time is very important, and what is to be done must be done quickly.

Diffusible stimulants may be given either hypodermically or *per rectum*.

Post-mortem Appearances. The mucous membrane of the tongue, mouth, throat and gullet, is commonly white, as if bleached, but it is sometimes coated with a portion of the brown mucous matter discharged from the stomach. This latter organ contains a dark brown mucous liquid, often acid, and having almost a gelatinous consistency. On removing the contents, the mucous membrane will be seen pale or black and softened, not always presenting marks of inflammation or abrasion, if death has taken place rapidly. It may, however, be coarsely corrugated and of a bright red colour. The mucous membrane is white, soft, and brittle, easily raised by the scalpel, and presents the appearance which we might suppose it would assume after having been boiled for some time in water. The small vessels are seen ramifying over the surface, filled with dark-coloured blood, apparently solidified within them. The lining membrane of the gullet presents the same characteristics. It is pale or dark, and appears as if it had been boiled in water, or digested in alcohol; it has been found strongly raised in longitudinal folds, interrupted by patches where the membrane has become abraded, and presenting a curious worm-eaten appearance. In a case which was fatal in eight hours the tongue was covered with white specks; the gullet was not inflamed, but the stomach was extensively destroyed, and had a gangrenous appearance. Portions of the mucous membrane were detached, exposing the muscular coat. With respect to the intestines, the upper portion may be found inflamed; but, unless the case is protracted, the appearances in the bowels are not strongly marked. In a well-marked instance of poisoning by this acid, however, which is recorded by Hildebrand, the mucous membrane of the stomach and duodenum was much reddened, although the patient, a girl of eighteen, died in three-quarters of an hour after taking one ounce of the acid, by mistake for Epsom salts.[1] In a case of poisoning in which two ounces of the acid had been taken, and death was rapid, the coats of the stomach presented almost the blackened appearance produced by sulphuric acid, owing to the colour of the altered blood pigment spread over them. In protracted cases, the gullet, stomach, and intestines have been found more or less congested or inflamed. In a case in which an ounce was swallowed, and death occurred on the fifth day, the stomach was slightly congested, and contained a bloody fluid, but the mucous membrane was entire.[2] Lesser found almost regularly in the stomach cloudy areas consisting of deposits of calcium oxalate, partly amorphous, partly crystalline.

[1] Casper's *Vierteljahrs-schr.*, 1853, Bd. 3, p. 256.
[2] *Lancet*, 1860, 2, p. 509.

In one instance the larynx was found filled with frothy mucus, and the left side of the heart and the lungs were gorged with dark-coloured fluid blood. In another, the appearance of sanguineous apoplexy were found in the brain. A person fell dead after retching violently. Apoplexy was supposed to be the cause of death. On an inspection of the body, it was found that a large cloth of blood was effused on the brain, and this appeared satisfactorily to account for death. But when the stomach was examined oxalic acid was detected in it. This poison had been taken, and had produced its usual effects. The deceased had taken it with suicidal intention, and the violent vomiting which it caused had led to death by apoplexy from effusion of blood.[1] Without a chemical investigation it is obvious that the real cause of death would have been in this instance overlooked. In a few cases there have been scarcely any morbid appearances produced by this poison.

The glairy contents of the stomach or its coats do not always indicate strong acidity until after they have been boiled in water. Oxalic acid does not appear to have a strongly corrosive action on the stomach. It is therefore rare to hear of the coats of the organ being perforated by it. In many experiments on animals, and in some few observations on the human subject, Taylor found nothing to bear out the view that perforation is a common effect of the action of this poison. The acid undoubtedly renders the mucous coat soft and brittle, and perforation of the coats may occur either during life or after death as a result of its chemical action. Wood has recorded the case of a female *æt.* 27, found dead, whose death had obviously been caused by oxalic acid, but the quantity taken and the duration of the case were unknown. The stomach presented, at its upper and fore part, near the cardiac opening, an irregular aperture of a size to admit the point of a finger. From this a dark gelatinous-looking matter, resembling coffee-grounds, was escaping in abundance. The perforation was enlarged during the removal, and presented the appearance of two large apertures separated by a narrow band. The stomach contained a bloody fluid, in which oxalic acid was detected, and the mucous membrane had an eroded appearance. The small intestines (jejunum and ileum) were similarly affected.

The kidneys show signs of acute nephritis, and on naked-eye examination a whitish zone due to the deposit of calcium oxalate may be seen in the cortex.

There are two features in the appearances found on autopsy which, if present, are very strongly corroborative of oxalic acid poisoning, practically pathognomonic. These are (1) the crystals in the stomach mentioned above and (2) similar crystals deposited in the urinary tubules, which on experimental grounds Kobert and Küssner regard as pathognomonic for oxalic acid poisoning. A. Lesser found calcium oxalate in the urinary tubules in a case in which death ensued in fifteen minutes after a dose of 15 grm. of oxalic acid.[2]

Analysis. So far as suspecting oxalic acid is concerned, Christison says: "If a patient, after swallowing a crystalline substance which tastes strongly acid, is seized almost immediately with violent vomiting,

[1] *Ibid.*, 1863, 1, 47.
[2] Kobert and Küssner, *Virch. Arch.*, 78, 209.

pain in the stomach, feeble pulse, cold sweats, and collapse and dies within half an hour, or even earlier, there can scarcely be a doubt that oxalic acid has been administered."

Chemical Analysis. In the Simple State. This acid may be met with either as a solid or in solution in water. It is soluble in about ten times its weight of cold water or two and a half times its weight of cold alcohol, but is only slightly soluble in ether. *Solid oxalic acid* crystallises in long slender prisms, which, when perfect, are four-sided. In this respect it differs from other common acids, mineral and vegetable. The crystals are unchangeable in air; they are soluble in water and in alcohol, forming strongly acid solutions. When heated on platinum-foil they melt, and are entirely dissipated without combustion and without being carbonised. Heated gently in a closed tube, they melt, and the vapour is condensed as a white crystalline sublimate in a cold part of the tube. The crystals are prismatic, like those obtained from the solution. There should be no residue whatever if the acid is pure; but the commercial acid generally leaves a slight residue of fixed impurity. By this effect of heat, oxalic acid is easily distinguished from those crystalline salts for which it has been sometimes fatally mistaken, namely, the sulphates of magnesium and zinc. These leave white residues in the form of anhydrous salts. Moreover, oxalic acid effervesces with sodium carbonate but gives no precipitate, while the two salts mentioned both give white precipitates with sodium carbonate but no effervescence. Half an ounce of the crystals is equivalent to about three teaspoonfuls.

When oxalic acid or an oxalate is heated with concentrated sulphuric acid, a mixture of carbon monoxide and carbon dioxide is evolved. The former burns with a blue flame, and, if passed into blood, produces the easily-detected carboxyhæmoglobin; the latter produces a milkiness when bubbled through lime water. (Test for CO_2 first).

Tests for oxalic acid in solution.

1. **Silver Nitrate Test.** Silver nitrate gives a voluminous white precipitate of silver oxalate when added to a solution of an oxalate or to a very dilute solution of oxalic acid. The precipitate is easily soluble in cold nitric acid (a reaction which distinguishes it from silver chloride). Since, in the case of free oxalic acid, silver nitrate liberates nitric acid ($C_2O_4H_2 + 2AgNO_3 = Ag_2C_2O_4 + 2HNO_3$), it is evidently desirable to carry out the test on a neutral solution. The acid solution of oxalic acid should therefore be neutralised by addition of ammonium hydroxide. (It should not, however, be made alkaline.)

If collected on a filter, thoroughly dried, and heated on thin platinum-foil, the precipitate of silver oxalate is dissipated with a slight detonation. When the oxalate is in small quantity, this detonation may be observed in detached particles on burning the filter, previously well dried.

2. **Calcium Test.** Any solution of a calcium salt gives a white precipitate of calcium oxalate with oxalic acids or an oxalate in solution. Since calcium oxalate is soluble in hydrochloric and sulphuric acids, though not in acetic acid, it is desirable to neutralise the test solution (as in the silver nitrate test). The identity of the precipitate should be confirmed by testing its solubility in acids and in ammonium hydroxide, in which calcium oxalate is insoluble. Dried and ignited on platinum

foil or porcelain, calcium oxalate is converted to calcium carbonate and thence to calcium oxide (quicklime), which gives a blue colour when placed on moist red litmus paper. Like other oxalates, too, it evolves carbon monoxide and carbon dioxide (without charring) when heated with concentrated sulphuric acid. Again like other oxalates, it decolourises potassium permanganate solution when dissolved in hot dilute sulphuric acid.

If it is warmed with a little dilute H_2SO_4, and if then potassium permanganate solution is added, drop by drop, to the hot liquid, the red colour is at first entirely discharged and continues to be so until the oxalate has been entirely oxidised, when a permanent pink colour denotes the end of the reaction. If the permanganate solution is of the usual standard strength (3·16 grammes per litre) and has been added from a graduated burette, we may now quickly calculate the quantity of oxalic acid, with quite sufficient accuracy for practical purposes, by remembering that 1 c.c. of the standard is decolourised, in these circumstances, by ·0063 gramme of crystallised oxalic acid.

3. **Lead Test.** Lead acetate, added to a solution of an oxalate or oxalic acid, gives a white precipitate of lead oxalate, soluble in cold nitric acid, but insoluble in acetic acid.

4. **Permanganate Test.** Oxalic acid or any oxalate, dissolved in hot dilute sulphuric acid (over 60° C., but not boiling), rapidly decolourises a solution of potassium permanganate.

Liquids containing Organic Matter. The process is the same whether it is applied to liquids in which the poison is administered, or to the *matters vomited*, or, lastly, to the *contents of the stomach*. This poison readily combines with albumen and gelatin, and it is not liable to be decomposed or precipitated by these or any other organic substances. It is, therefore, commonly found in solution in the liquid portion, which will then be more or less acid. Preliminary tests may be made on this fluid direct—the calcium test, or addition of copper sulphate after boiling to remove protein. If oxalic acid is present in moderate amounts, copper sulphate will give a greenish-white precipitate.

Oxalic acid, like many other crystalloids may be partly separated from colloid organic matter by dialysis. The stomach contents (with the minced stomach, if necessary) or other material is placed in a parchment or collodion sac which is suspended in distilled water. The oxalic acid, with other crystalloids, passes into the water and to this solution, after concentration by evaporation, the appropriate tests may be applied. Instead of distilled water, a solution of calcium acetate or sulphate may be used, and in this case the oxalic acid, dialysing into the solution, gives a white precipitate of calcium oxalate- often as octahedral crystals: this procedure should only be used for part of the material, so that confirmatory tests may be applied to the remainder.

An alternative procedure, much better for quantitative work, is as follows. Boil the liquid to be tested, filter, wash the precipitate with distilled water and, to the combined filtrate and washings acidified with acetic acid, add lead acetate solution until no further precipitation occurs. Filter or centrifuge off the precipitate (which contains all the oxalic acid as lead oxalate), wash, suspend it in a little distilled water, and treat it with hydrogen sulphide for several hours. This will form black lead sulphide and liberate the oxalic acid which will pass into solution. Filter off the precipitate, wash, and concentrate the combined

filtrate and washings by evaporation, in which process the excess hydrogen sulphide will be removed. Oxalic acid can then be identified in the solution by the usual means, and in an aliquot part the amount of the acid can be determined—by precipitating as calcium oxalate and either weighing the precipitate or dissolving in hot dilute sulphuric acid and titrating with a standard solution of potassium permanganate.

Sometimes the chemical evidence may depend on *stains* on articles of *clothing*. Oxalic acid discharges the colour of some dyes, and slowly reddens others; but unless the stuff has been washed, the acid remains in the fabric and may be detected there. It does not corrode nor destroy the stuff so readily as mineral acids.

In cases of poisoning, the residual quantity found in the stomach is generally small. In one case, in which about an ounce and a half had been taken and in which the patient died in two hours, only thirteen grains were recovered. In a case which occurred at Bristol in 1868, a woman took upwards of three-quarters of an ounce of oxalic acid (360 grains), and died in ten minutes. It is stated that not more than two grains were obtained from the coats of the stomach. The vomiting had been violent, and the greater part of the poison had been thus ejected. It seems that the woman had vomited into a pail containing calcareous water, and it was observed that this water acquired a milky white appearance, owing to the action of the acid on the salts of calcium.[1] In *R.* v. *Cochrane*,[2] in which it was charged that two children, aged six and four years respectively, had been wilfully poisoned by their mother, it was stated by the medical witness, Edwards, that he found forty-two grains of oxalic acid in the stomach of the elder, and twenty grains in that of the younger child. It was not clearly established when or how this large quantity of poison could have been wilfully administered to the children, and the prisoner was acquitted.

Since the soluble oxalates give the same reactions as oxalic acid itself, to prove the presence of oxalic acid itself, it will be necessary to ascertain the presence of an oxalate, and also to determine the amount of free acid present in the solution submitted to analysis, by titration with an alkali (showing, also, that no other free acid is also present). Since, however, the soluble oxalates are themselves highly poisonous, in practical toxicology it is not always necessary to do this in order to prove that a poison has been taken.

Poisoning by Oxalates

Source and Method of Occurrence. Binoxalate of potash, also called salts of sorrel or salts of lemon, is a commercial product used largely in straw bleaching, etc. Workpeople frequently keep a solution of it in ginger-beer bottles, etc., and drink it by mistake. Accident is thus the common form of poisoning by this salt. The crystals are sometimes mistaken for Epsom salts.

Toxicity and Fatal Dose. The soluble oxalates destroy life almost as rapidly as oxalic acid itself. In one case, half an ounce of salts of sorrel killed an adult in so short a time as *eight minutes*; but probably the fatal effects were in this instance accelerated by the debilitated state

[1] *Chem. News*, April 24th, 1868, p. 205; and *Pharm. Jour.*, May 1868, p. 543.
[2] Liverpool Sum. Ass., 1857.

of the person who took it. In another case reported by Chevallier, death took place in ten minutes.[1] In one instance in which it was supplied by mistake for Epsom salts, it caused death in an hour and a half.[2] In a reported case,[3] death appears to have been caused by this salt as the result of chronic poisoning. Out of nine recorded cases of poisoning by this substance, six proved fatal, while in three the patients recovered.

Duration and Symptoms. In this respect the oxalates resemble very closely the acid itself, except that the irritant action may be less marked, and the symptoms of its depressant effect on the heart after absorption are more marked.

In a case of recovery, a young lady, aged twenty, swallowed an ounce of the salt dissolved in warm water. She was not seen by any one for an hour and a half; she was then found on the floor, faint and exhausted, having previously vomited considerably. There was great depression, the skin was cold and clammy, the pulse feeble, and there was a scalding sensation in the throat and stomach, with continued shivering. Proper medical treatment was adopted, and she recovered in two days, but still suffered from debility and great irritation of the stomach. During the state of depression it was remarked that the conjunctivæ of the eyes were much injected, and the pupils dilated. There was also great dimness of vision.[4]

Treatment. The same as for the acid, except that the stomach-tube may be used, as there is less fear of corrosion.

Post-mortem Appearances. In the chronic case mentioned above a girl was charged with the murder of her father. He began to be ill about December 5th, and he died on January 26th following. He suffered from vomiting, heat and irritation in the mouth and throat, prostration of strength, and constant pains in the chest and abdomen. After death the appearances were—inflammation of the mucous membrane of the stomach and part of the bowels. They contained a dark-coloured fluid. The mucous membrane of the gullet was destroyed. The coats of the stomach, which were thickened and injected, had a gangrenous appearance. There was no proof that the prisoner had had possession of the poison until January 11th, five weeks after the symptoms had begun in the deceased. The symptoms before and subsequently to this date were similar. The absence of proof of possession led to the acquittal of the prisoner; still it would be difficult to account for the symptoms and appearances on any theory of disease.

Analysis. This salt is not very soluble in cold water, but its solution may be readily mistaken for that of oxalic acid. It is not dissolved by alcohol; and this distinguishes it from oxalic acid. (1) The aqueous solution has an acid reaction; and (2) it gives a precipitate both with nitrate of silver and sulphate of calcium, like oxalic acid; but with the latter reagent the precipitation is much more copious than in the case of oxalic acid itself. It is further distinguished by its *crystals*, which, when slowly produced on a glass slide, assume the shape of small

[1] "Ann. d' Hyg.," 1850, 1, 162.
[2] *Pharm. Jour.*, 1873, p. 760.
[3] *Edin. Month. Jour.*, July 1862, p. 93.
[4] *Med. Gaz.*, vol. 27, p. 480.

rhombic prisms, often grouped in a plumose form, and by *heating* a portion on platinum-foil. While oxalic acid is entirely volatile, the binoxalate leaves an ash, which, when sufficiently heated, is white and alkaline; it may be proved to contain carbonate of potassium by its dissolving with effervescence in diluted nitric acid, and forming potassium nitrate.

In some instances this poisonous salt has been supplied by mistake for cream of tartar, and has caused death. Cream of tartar, or acid-tartrate of potassium, leaves a black alkaline residue when heated in closed vessels (*i.e.*, in a very limited supply of oxygen). Its solution is less acid than that of the salt of sorrel. It is not precipitated by nitrate of silver or sulphate of calcium. It chars when heated with concentrated sulphuric acid. The different action of the two salts on writing-ink affords a simple means of identification.

Cases. In March, 1894, an inquest was held at St. Helens, Lancashire, on a man who "took something out of a cup" and died in less than half an hour. The contents of the stomach responded to the tests for oxalic acid, and enormous quantities of oxalate crystals were found in the urine, but there was no erosion, not even inflammation of the stomach nor of any part of the alimentary tract. There was no evidence to show how or whence the acid was obtained, and, except for the rapidity of death, the case was doubtfully one of oxalic acid poisoning. It seems most probable that there was an accidental mixing of oxalic acid with Epsom salts, which deceased thought he was taking. The dose was taken on an empty stomach early in the morning, which probably accounts for the rapid death. The quantity swallowed was quite problematical.

Dr. Hale White showed before the Pathological Society in March 1896, two cases of acute nephritis produced by oxalic acid poisoning.

In *R. v. Morris*[1] it was proved that the prisoner had attempted to administer a liquid poison forcibly to her daughter, a girl aged six years. The liquid was sour in taste, made the girl's lips smart, and caused vomiting. There was dryness of the lips, and inflammation of the lining membrane of the mouth. No portion of the substance administered could be procured, but a crystalline deposit of oxalic acid was obtained from some stains on the dress of the child. The woman was convicted.

White published a report of a case of poisoning with oxalic acid in which the symptoms and appearances are contrasted with those caused by disease, and compared with those usually assigned to oxalic acid. The poison was not detected in the contents of the stomach, but the sheets on which the patient had vomited yielded one or two grains of oxalic acid. The patient lived forty hours after vomiting had set in.[2]

Poisoning by Carbolic Acid or Phenol (C_6H_5OH)

Source and Method of Occurrence. Since the discovery of its antiseptic properties carbolic acid has been manufactured in enormous quantities from coal tar. The pure acid is a white crystalline body, consisting of colourless interlaced needles, but on exposure to light and air, it frequently acquires a pinkish colour. The crystals melt at 40–43°C. It has a characteristic, and not unpleasant, odour. When a small proportion of water is added to the crystals, they liquefy. It is moderately soluble in water (100 c.c. of saturated solution at the ordinary room temperature contain about 10 grammes of phenol). The commercial crystals have an unpleasant odour, which is due to impurities: and they acquire a red colour on exposure to light. They melt at about

[1] C. C. C., December 1866.
[2] *Boston Med. and Surg. Jour.*, January 27th, 1870.

91°F. (26°C.). More commonly carbolic acid is met with either as a colourless or more or less light brown liquid, of peculiar odour, containing about 80 per cent. of phenol, or as a dark brown liquid chiefly consisting of heavy oil of tar with about 30 per cent. of the acid. These are largely used as disinfectants. Carbolic acid does not redden litmus paper. Deaths from the internal use of carbolic acid—usually accidental or suicidal—are common. In England and Wales, within recent years, (1931-1940) the annual average number of suicides by means of carbolic acid has been about forty, while the average number of accidental deaths from the same cause is between two and three. The corresponding figures over the same period for lysol and other phenol and cresol derivatives are much higher *viz.* 275 suicidal and 6 accidental deaths. These substances are used in about half of the cases of suicide from all causes excluding coal gas.

Toxicity and Fatal Dose. The official dose of the acid is one to three grains, so that *per se* it does not deserve to be ranked amongst the very dangerous poisons. It is its universal use as a domestic disinfectant, and the ease with which it can be obtained in large quantities, that constitute its danger. There is reason to believe that if absorbed, a few grains of the poison might prove fatal, and eighty grains have killed an adult. Two fluid drachms of the liquid acid have killed a child two years of age in twelve hours.[1] In one case a child, six months old, was killed by the administration of a **quarter of a teaspoonful** of the acid dissolved in glycerine—one part of acid to five of glycerine.[2] Recovery has, however, taken place after large doses. A girl, *æt.* 14, swallowed six fluid drachms of the undiluted (? 30 per cent.) acid. In twenty minutes she was comatose and breathing stertorously; her face was livid, the pulse was small and irregular; the pupils contracted, but not so much as in opium poisoning. The stomach was washed out with soap and water, and then with milk and water. In an hour, the lividity diminished, and consciousness slowly returned. She did not complain of any gastric irritation.[3]

One cause of the danger of phenol is that it is rapidly absorbed, not only from the alimentary tract, but from the rectum, vagina, serous cavities, wounds, and even through the intact skin.

Duration. The symptoms usually come on immediately when the acid has been swallowed in poisonous amounts. Death has occurred almost immediately, **within three minutes,** and several times within twenty minutes of the administration of the poison. Littlejohn reports a death in fifteen to twenty minutes.[4] It usually supervenes within four hours. Occasionally, in fatal cases, life may be prolonged for twenty-four and even forty-eight hours. Rawlings[5] reports a case of death after seven days.

Reimann[6] reports the death of a newly born infant within a few hours from the application of pure phenol to the navel.

[1] Guy's Hosp. Rep., 1867, p. 233.
[2] *B.M.J.*, May 20th, 1882.
[3] *Ibid.*, 1882, 1, p. 939.
[4] *Lancet*, 1900, 2, p. 176.
[5] *St. Bartholomew's Hosp. Jour.*, 1896-97, 4, 103.
[6] *Verteljahrssch. f. gen. med.*, 1891, 3 F, 2, 63.

Modica[1] gives an account of the death of a girl four years of age who died in four hours after receiving an enema containing carbolic acid. In a similar case[2] 144 grains diluted with water injected into the bowel of a boy caused death in fourteen hours. Cases are also recorded of poisoning from inhalation of the fumes and from absorption from the unbroken skin.

Symptoms. As with so many other poisons, there are two classes of cases in carbolic poisoning:—1. The acute form; by far the most common, 2. The subacute or chonic. The latter might perhaps be better styled the untoward effects of carbolic acid when employed for medical purposes, rarely seen nowadays, since the advent of more modern antiseptics.

In the Acute Form. When the poison is swallowed in solution, or in the form of an undiluted liquid, the patient experiences a hot burning sensation, extending from the mouth to the stomach. This feeling is experienced during the act of swallowing: and the lining membrane of the mouth is white and hardened. Carbolic acid is rapidly absorbed, and in the course of a few minutes may produce profound effects. In many instances the rapidity of action is comparable to that of prussic acid. Although the local application of the acid to a part is commonly followed by severe burning pain, this may be entirely absent and even a local diminution of sensibility may be produced. This and also the fact that vomiting is often absent is due to the fact that carbolic acid rapidly anæsthetises the part to which it is applied. Nervous symptoms are those most strikingly manifested, such as delirium, giddiness, and profound insensibility. Nausea and vomiting are present in not more than one-fifth of the observed cases, but may be severe and uncontrollable. When vomiting occurs there is an additional danger in that the poison may be aspirated into the lungs, thus causing bronchitis and broncho-pneumonia. Lung symptoms and broncho-pneumonia may also develop after ordinary ingestion. The respiration is slow and stertorous. There is extreme feebleness of the pulse, and dry harsh skin, with lividity of the surface. The pupils are generally minutely contracted. This state of the pupils and the associated coma may lead to a suspicion of opium poisoning. Convulsions and trismus are not infrequently observed. The blood-pressure is usually low and the scanty urine contains albumin and free hæmoglobin.

In the subacute or chronic form the symptom that arrests attention is the peculiar green discoloration of the urine. This was first pointed out by Sir Thomas Stevenson in 1868, and owing to the prevalent use of the acid in the early 'eighties for the dressing of wounds, it was soon observed by many surgeons. It was a danger signal and commonly disappeared with the discontinuance of the use of the acid. It is, however, often observed in cases that prove fatal. It was commonly the only symptom in ordinary surgical practice, but sometimes more serious features were added, such as giddiness or drowsiness. The urine when voided is often of normal colour or with a slightly green tint. On exposure it gradually darkens to an olive green or black. In the body, phenol is partly oxidised to hydroquinone and pyrocatechol. These

[1] *Gaz. d. Osp.*, 1907, 28, 1540.
[2] *Lancet*, 1883.

substances along with unchanged phenol are then excreted, partly free, and partly in unstable combination with sulphuric and glucuronic acids. The further oxidation of hydroquinone and pyrocatechol in the voided urine is the cause of the darkening. In some cases the withdrawal of sulphuric acid may cause a marked diminution in the blood sulphate. Blood and casts may also be present from damage to the urinary tubules. Hæmoglobinuria and jaundice may occur from the hæmolytic action of the drug.

A case of carbolic acid poisoning can rarely fail to be recognised. In the acute cases the whitening of the mouth, the brown eschars which form on the skin at its angles where the poison trickles from the mouth, the odour of the breath, and the profound insensibility and stertorous breathing, with minutely contracted pupils, rarely leave any doubt as to the nature of the case. Sir Thomas Stevenson has, nevertheless, met with a case in which a pure form of the acid having been taken for suicidal purposes, the odour of carbolic acid escaped notice. In the chronic cases the green urine is the characteristic feature which cannot escape observation. Many deaths have occurred from the external use of the acid, and its use as an antiseptic lotion has repeatedly produced serious, if not fatal, results.

A well-known local lesion following the application of phenol in ordinary or even feeble concentrations is gangrene, which is usually met with in the fingers and in wounds, but has also been seen in the genital organs.

Treatment. The corrosive action of carbolic acid is not very marked, and therefore a soft stomach tube may generally be used with care, but it must be remembered that the anæsthetising action of the acid may prevent the operator knowing that the tube is doing damage. After emptying, the stomach should be well washed out with lukewarm water, in which some magnesium sulphate, or saccharated lime, may with advantage be dissolved, in order to afford an opportunity for the phenol to combine and form an innocuous ether-sulphate. Dixon[1] states that intravenous injection of a solution of sodium sulphate, or better persulphate, gives relief; the respiration recovers, and the blood-pressure rises, although recovery is slow. White of eggs and milk or adsorptive charcoal (*e.g.*, Carbo medicinalis, Merck.) may be given, but should be removed after a very short time—*i.e.*, they are to be used for gastric lavage. Olive oil has been recommended, but with doubtful advantage. Several observers have noticed that apomorphine failed to produce emesis in phenol poisoning. Kolar[2] states that atropine sulphate is of great value as an antidote. It possibly stimulates the respiratory centre. External warmth, with stimulants such as ether administered hypodermically, or alcohol by the mouth or rectum, have been recommended. Alcohol by the mouth is claimed to prevent the cauterising action of the phenol, but its use has been condemned by some observers.[3] Liquid paraffin has been recommended for a similar purpose, and since it is absorbed only very slightly by the mucous membranes it may be valuable in restricting the caustic action of phenol. If respiratory paralysis

[1] *Manual of Pharmacology*, 1925.
[2] *Jour. Amer. Med. Ass.*, 1897.
[3] *Johns Hop. Hosp. Bull.*, 1915, 26, 98.

appears imminent, artificial respiration with administration of oxygen and carbon dioxide should be employed.

Post-mortem Appearances. Stains produced by the poison may be present at the angles of the mouth and on the chin, and its odour may be perceptible. The mucous membrane of the mouth may be softened, and either white or ash-grey in colour, that of the œsophagus being similarly affected in parts; on account of the shorter period of contact, the changes in the mouth and œsophagus are not usually so well marked as those in the stomach. The peritoneal surface of the stomach may be injected, its mucous coat usually being corrugated, toughened, and of a brown colour; in parts it sometimes appears stiff and leathery as though it had been tanned; in other instances it is softened and easily detached. It has been observed to be of an ash-grey colour with small hæmorrhagic points; actual erosion is uncommon. Blood-stained mucus has been found in the stomach. The duodenum may present a similar appearance, the brown colour being sometimes limited to the summit of the valvulæ conniventes or sometimes seen in the form of a series of parallel brown lines running across the bowel for fully twelve inches. The acid may transude through the walls of the stomach and cause staining and hardening of the liver and spleen where they come into contact with it.

The lungs are frequently congested and sometimes œdematous. The kidneys may show acute nephritis.

Analysis. The characteristic smell of phenol in the viscera is the most valuable indication of its presence. To detect phenol in viscera, vomitus, etc., the material is thoroughly mixed with dilute (5 per cent.) sulphuric acid in sufficient quantity to make it distinctly acid. The mixture is then distilled in a current of steam until the distillate no longer gives a red colour when boiled with Millon's reagent. The distillate is extracted first with petrol ether (in which phenol is almost insoluble) and then with ether (in which it is very soluble). The ethereal extract is shaken with sodium bicarbonate solution, which removes any salicylic acid present (salicylic acid is a derivative of carbolic acid, and responds to many of the tests used for the latter). The ethereal extract is then dried by shaking with anhydrous sodium sulphate and evaporated. The residue may be dissolved in water, and submitted to various tests, but it is well to remember that very small amounts of (combined) carbolic acid exist normally in the body, and that in disease, greater quantities may be present.

Tests.

1. **Million's Test** (given also by nitrophenols and by proteins). A red colour is produced when a dilute solution of phenol (even 1 : 2,000,000) is warmed with Millon's reagent. The reagent is prepared by dissolving mercury in an equal weight of 63 per cent. nitric acid (warming to start the action) and diluting the resulting solution with its own volume of water.

2. **Ferric Chloride.** A drop of ferric chloride gives an intense blue-violet colour with phenol. The colour is diminished in intensity by mineral acids, ammonia, or excess of ferric chloride. Alcohol prevents its development.

3. **Bleaching Powder.** A solution of phenol, made alkaline with a quarter of its volume of ammonia, develops a transient blue colour on addition of a few drops of bleaching powder (calcium hypochlorite) solution or when shaken with bromine vapour. The colour becomes permanent on addition of more hypochlorite.

4. **Sodium Nitrite.** Add a drop of 10 per cent. sodium nitrite solution to 10 c.c. of the solution to be tested. Pour the mixture carefully over concentrated sulphuric acid so that there is no mixing and two layers are formed. In the presence of phenol, a double ring appears at the junction of the layers—emerald green below, and ruby red above.

5. **Bromine.** Even in very dilute solution, phenol yields a copious precipitate of tribromophenol, when an excess of bromine-water is added. These crystals have a characteristic form (fine stars or needles) when examined under the microscope. The precipitate when washed on a filter, and treated in alcoholic solution with sodium amalgam, again yields phenol (carbolic acid). Tribromophenol melts at 95° C., is insoluble in water or acids, but is soluble in alkalis, alcohol, and ether.

Cases. In dealing with such a common poison it is needless to quote cases unless they present unusual features.

Early in 1904 Dr. F. J. Smith saw, in consultation, a small Jewish baby suffering from carboluria, the effect of the external application of carbolic lotion after circumcision. The child died within twenty-four hours of his visit; there were no pathological changes discovered on autopsy visible to the naked eye, but the condition of the urine was unmistakable. It is probable that this accident occurs more commonly than is suspected, for other such cases have been communicated. See *Lancet*, 1904, 1, p. 1279.

A robust woman, *æt.* 30, swallowed nearly half an ounce of an alcoholic solution of carbolic acid, containing 35·8 per cent. of the poison. It was ascertained by the use of the stomach-pump that rather more than half of the poison was removed from the stomach, but that at least ninety-two grains of the poison must have remained for absorption. The most prominent symptoms were insensibility within ten minutes and dizziness speedily passing into profound coma, irregular breathing and pulse, contracted pupils, extreme blueness (cyanosis) of the surface of the body, depression of the body temperature to 94° F., and hæmoglobinuria, *i.e.*, the presence of unaltered blood-pigment in the urine, which contained no red blood-corpuscles. This last condition set in one hour after the poison was taken, and lasted for seven hours and a half. The urine gave the reaction for carbolic acid for two days. The woman recovered.[1]

Poisoning by Cresol

Cresol poisoning is almost invariably due to one or other of the proprietary "coal tar" disinfectants, such as **Creolin, Lysol, Cyllin, Izal, Jeyes' Fluid, Trikresol, Kerol, etc.**, which contain cresols and other phenols frequently held in solution by soap.

Of the three isomeric cresols (hydroxy-toluene) meta-cresol is stated to be the least toxic (rather less so than carbolic acid), and para-cresol the most toxic. The symptoms are similar to those of poisoning by carbolic acid.

(1) Poisoning by Creolin

Source and Method of Occurrence. This substance is thus described in the "Extra Pharmacopœia": "Creolin, a dark-coloured liquid antiseptic; said to contain 20 per cent. cresylic acid, with neutral hydrocarbon oil and resin soap."

[1] *Berlin Klin. Woch.*, 1881, No. 48.

" Dr. Pinner, physician to the Jewish Hospital in Hamburg, reports the case of a woman sixty years of age who had attempted suicide by swallowing seventy-five grammes of creolin. It was at first not known which of the preparations called creolin had been taken, but the druggist from whom it was purchased subsequently stated that he only sold Pearson's. The woman was found unconscious in her room, and was taken to the hospital two hours after the attempt on her life. She was then comatose, with livid face, moist skin, and blue lips; her right pupil was larger than the left, and there was no reflex action of the cornea. Her temperature was 36·6° C., her pulse 112 and regular, and her respiration deep and snoring, with tracheal rales. In the pharynx there was a large quantity of white mucus: the palatal arches and the posterior wall of the palate were covered by a grey superficial coating. There was a strong smell of creolin in the breath. By the stomach-pump three hundred grammes of a thick grey fluid mixed with mucus were withdrawn, and pressure on the abdomen caused thick brown drops of pure creolin to come through the tube. The first urine (one hundred and fifty grammes) was of a light yellow colour, without albumen, sugar, or indican; the bromine-water test showed traces of tar acid in it. Microscopical examination revealed nothing abnormal. Milk given through an œsophageal tube was immediately vomited, but was eventually retained. An hour after admission into the hospital there were well-marked symptoms of shock, for which injections of ether were successfully made. There was also copious diarrhœa, and the stools had an odour of creolin. Some hours later the patient regained consciousness and afterwards became better. She complained of severe pain in the epigastric region and in the pharynx. The urine (eighty grammes) was now of a dark-green colour, had an odour of creolin, and contained traces of albumen; by the tribromophenol reaction it was estimated that 7·5 c.c. of tar substances were present in 100 c.c. There was no haemoglobinuria, and only a small quantity of leucocytes and red corpuscles and some casts. The patient slept very well during the first night, and on the following day there was an obvious improvement, the pulse, temperature, and respiration being normal, although she still complained of headache, want of appetite, and hoarseness; erosions on the epiglottis and the arytenoid cartilage were observed with the laryngoscope. On the fourth day there was more diarrhœa, the stools still smelling of tar products. The urine continued for a week to be of a green colour, and contained $\frac{1}{2}$ per cent. of albumen, with much indican. After four weeks the patient was able to leave the hospital in perfect health, and did not show any trace of injury from the poisoning. Dr. Pinner states that this is the seventh case of creolin poisoning described in medical literature, and that it was remarkable for the extremely severe symptoms which appeared almost immediately after the poison had been swallowed. Creolin, he says, used to be considered harmless because it could not be absorbed by the stomach, but in the above case the gastric juice very soon transmuted the creolin into a form which very quickly passed into the circulation and appeared after two hours in the urine. It was very remarkable that the urine of the first four days, even when kept in open glasses, showed neither any growth of bacteria nor any putrefaction, and the normal development of bacteria after exposure to the atmosphere occurred only in the urine passed on and after the fifth day. Dr. Pinner, therefore, suggests that the internal use of creolin may, perhaps, be useful in bacterial diseases of the genito-urinary system, such as pyonephrosis, cystitis, etc. As it also checked fermentation in the bowels, perhaps intestinal derangements attended by extreme fermentation may be successfully treated by creolin. Notwithstanding the alarming symptoms of the first few hours, the patient very soon recovered, the creolin being obviously removed by the kidneys, which, though disordered for a short time, did not sustain any permanent injury. Creolin, therefore, according to Dr. Pinner, must be regarded as a comparatively innocuous substance. Another case of alleged creolin poisoning is reported by Dr. Gras, of Vilshofen, in the *Blätter für Gerichtliche Medicin*. A tuberculous woman, thirty-five years of age, was treated on the sixth day after confinement with vaginal irrigations of 3 per cent. creolin solution on account of fœtid lochia. After the third irrigation the patient suddenly became collapsed and lost consciousness, the extremities being cold, the pulse feeble, and the respiration shallow; but injections of camphor and other restoratives were very soon followed by recovery, and no traces of carbolic acid were found in the urine. It

seems rather doubtful whether these symptoms were due to creolin poisoning or were merely the effect of shock occurring in a woman weakened by child-birth and tuberculosis. There were no positive toxic symptoms, and the quick recovery makes it probable that the seizure was not really due to poisoning."[1]

Analysis. Separation from the viscera, etc., is achieved, as in the case of phenol, by acidification and steam distillation. Cresols are extracted from the distillate by petroleum ether (in which phenol is almost insoluble).

Cresols give a blue colour with ferric chloride (best in neutral solution), but on addition of bromine water they form **liquid** tribromo-cresols.

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(2) Poisoning by Lysol

Lysol is a dark-coloured alkaline liquid consisting of mixed cresols, the homologues of phenol, dissolved in soap solution.

Toxicity and Fatal Dose. The relative toxicities of carbolic acid and lysol are usually placed at eight to one. Thus, if ten grains is the lethal dose of the former, eighty grains would be that of the latter.

Cases. "Some two months ago a child aged three weeks, whose father had given it lysol instead of a laxative, was admitted to the Foundling Hospital suffering from paroxysms of cough and dyspnœa. The child died next day, and the *post-mortem* examination, made by Professor Hoffmann, gave the following results. The mucous membrane of the lips was greyish, and could be stripped off; the epithelium of the tongue and the mucous membrane of the larynx and trachea were swollen; the left lung was consolidated, violet-blue in colour, and injected with blood; the mucous membrane of the anterior part of the stomach was pale rose-coloured, whereas on the posterior part hyperæmia and tumescence were observed. Dr. Haberda, assistant to Professor Hoffmann, has shown that the poisonous action of lysol is due to its containing cresols. It cauterises the skin and mucous membrane, and when absorbed it affects the brain and spinal cord, producing unconsciousness, general spasms, reduction of temperature, and bleeding into the uriniferous tubules. He therefore recommends that it should be prescribed only in dilute solution."[2]

The following is from the *B.M.J.*, 1900, 2, p. 1498 :—

"On September 10th, I was called to one of the schools here to see a European boy, aged fourteen, who had been suffering from 'dysentery' (?) for about one month. At 1 p.m. he had taken an injection of rather less than one ounce and a half of lysol in about a pint of water, using an ordinary Higginson's syringe. At 1.30 p.m. he was found in bed quite unconscious, in which state he remained until my visit at 5.15 p.m.

"He was then in a state of complete collapse, perfectly unconscious, could not be roused at all, lying partly on his side, with the legs drawn up, with pinhole pupils. He was sweating, the pulse was almost uncountable and running, and the breathing rapid, but not abdominal, forty per minute. The temperature was subnormal. The heart and respiratory sounds were normal. The abdomen was not distended and only slightly tense; there was apparently no tenderness on pressure. There was no carbolic smell from the breath or sweat. The anal orifice was slightly discoloured from the action of the lysol.

[1] *Lancet*, 2, 1895, p. 1178.
[2] *Lancet*, 1, 1895, p. 1221.

"The boy was evidently dying. I gave hypodermic injections of strychnine and of ether, which had some slight effect on the pulse. I also washed out the bowel with warm water, which was at first retained, but, on making slight pressure over the abdomen, came away quite uncoloured, but containing numerous white shreds like curds.

"The boy had not been disturbed, when suddenly a quantity (two ounces or so) of dark brown grumous blood was ejected from the mouth and nose (there was no excoriation or discoloration of the mouth), and in five minutes (5.45 p.m.) he was dead. No *post-mortem* examination was made.

"I have frequently in similar cases (really lower intestinal catarrh) used irrigations with the long soft tube of half an ounce of Jeyes' fluid (creolin) in three pints of warm water, which has been evacuated, and afterwards I have left in, for from two to six hours, a drachm to a drachm and a half of Jeyes' fluid in eight ounces of water without ever having had the slightest ill effect, and almost invariably with marked benefit. Boric acid used in a similar manner often causes severe headache and bilious vomiting. Lysol is evidently more dangerous." (Hartigan.)

Vide also for fatal cases in children *B.M.J.*, Epit., 1898, 2, p. 63, and *B.M.J.*, Epit., 1901, 2, p. 73.

By mistake a nurse gave a woman, *æt.* 38, suffering from typhoid, some twelve grains of lysol, of which she swallowed ten grains. Five minutes later the patient was markedly cyanosed, and in a state of stupor. The breathing was noisy, difficult, quickened, and irregular. Spasm was at times noted in the extended arms. The pulse could not be felt. Camphor was injected, and the stomach washed out a few minutes later. The wash-water smelt of lysol. The heart's action stood at 160 per minute, and was a little irregular. The pupils were somewhat dilated, and did not react to light. The tongue was dry. The patient began to improve after the camphor injections, but she was still unconscious two hours after taking the poison. She woke up three and a half hours later, and complained of burning in the mouth. Milk and natrium sulphuricum were given by the mouth. She vomited a little later, and the vomit smelt of lysol. The urine contained albumen and casts. The patient ultimately recovered after passing through a further relapse of the typhoid fever. The local symptoms here were not severe. The danger lay in the general symptoms, which consisted of rapid coma and cardiac weakness. The temperature fell rapidly from 39° to 36° C. The symptoms resembled those of carbolic acid poisoning. It is usually said that the difference between lysol and carbolic acid poisoning is that in the former the symptoms do not come on for some little time, but here they appeared in ten minutes; hence no generalisation can be made. The typhoid fever may have accounted for the severity and early appearance of the symptoms. Thus, as with carbolic acid, the symptoms of lysol poisoning may not correspond with the size of the dose.

Analysis. See p. 393 under "Creolin." The tests for cresols do not identify the poison as "Lysol," but only as one of the many disinfectants containing these substances. The choice is narrowed somewhat if fatty acids are also detected and shown to be derived from the poison.

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Poisoning by Creosote

Source and Method of Occurrence. It is a product of the dry distillation of wood, and is extracted from the wood tar thus obtained. It consists of a mixture of guaiacol, creosol, and other phenols. It has some reputation as an antiseptic for internal use, and also for the treatment

of phthisis. It is a powerful drug with local effects similar to those of carbolic acid, but is not commonly used in present-day medical practice.

The official dose is two to ten minims, cautiously increased.

Taylor records in previous editions that ten drachms of crude "oil of tar" caused the death of a gentleman in 1832, and he states that this was due to the creosote contained in the dose.

Treatment. As for carbolic acid, *q.v.* p. 318.

Analysis. Separation from organic admixture is to be effected as is directed for phenol.

Creosote may be recognised by its odour. In alcoholic solution it may be distinguished from phenol by adding a few drops of a solution of ferric chloride; a green colour is produced, which disappears on dilution with water. Phenol similarly treated gives a lilac colour which does not disappear on the addition of water.

The guaiacol may be detected (in the distillate) by the development of a blue colour on addition of hydrogen peroxide and a drop or two of blood.

Cases. "In the *Intercolonial Medical Journal of Australasia* of October 20th, 1900, Mr. Herbert M. Hewlett relates the following case of creosote poisoning in a child. A girl, aged three and a half years, swallowed about two drachms of creosote. Immediately she complained of abdominal pain and was given a tablespoonful of olive oil. She rapidly became unconscious. About twenty minutes afterwards, when she was seen by Mr. Hewlett, she was insensible, with pale face, cyanosed lips, shallow respiration, and imperceptible pulse. The eyes were fixed, the pupils were contracted and immobile, the extremities were cold, and the muscles were flaccid. The stomach was washed out with two quarts of warm water. A small quantity of food was present, and the washings smelt strongly of creosote. The stomach was next washed out with two pints of a strong solution of magnesium sulphate, and about six ounces were left in the viscus. Strychnine (one-twenty-fifth of a grain) was injected hypodermically, and she was put in blankets and surrounded by hot bottles. An enema of Liebig's extract and brandy was also given. About eight minutes after emptying the stomach the colour of the lips improved, and the pulse became perceptible. Ten minutes later the colour was good, and the respiration was full and regular. The pupils were still contracted, but reacted to light, and the extremities were warm. In a couple of hours she drank white of egg and milk, but complained of pain over the stomach, which was relieved by hot fomentations. Urine passed many hours after taking the drug was dark brownish. The first motion was also dark and smelt strongly of creosote. A second sample of urine was of a watery green colour. No after-effects followed, and in a couple of days the child was running about again. Creosote closely resembles carbolic acid in its action, but is a weaker caustic, and does not produce convulsions."[1]

In the *B.M.J.*, 1898, 1, p. 144, a case of extraordinary tolerance to creosote is recorded. The patient began with one drop three times a day. This was rapidly pushed till he was taking 340 drops a day, and for three months he continued to take from three to four drachms daily.

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Stevenson, "Poisoning by Creosote." Guy's Hosp. Rep., 1875, 20, 150.

[1] *Lancet*, 1, 1901, p. 120.

Poisoning by Pyrogallic Acid (Pyrogallol)

1.2.3. Trihydroxy-benzene, $C_6H_5(OH)_3$

Source and Method of Occurrence. The acid is largely used for the purposes of photography. Very few cases of poisoning by it are recorded for human beings, but, according to Personne, it operates powerfully on animals. Two healthy dogs were selected, and into the stomach of one a dose of two grains of pyrogallol dissolved in water was injected; and twice this quantity was administered to the other dog. The animals died after fifty and sixty hours respectively. The symptoms are said to have resembled those of phosphorus poisoning, and after death the muscular tissue of the heart was found in each case to have undergone fatty degeneration. The acid causes shrinkage of the red blood cells, and the hæmoglobin escapes to the plasma, where it is converted to methæmoglobin. There may, therefore, be intense cyanosis. Icterus follows, and the urine contains blood and albumen. The urine is dark brown in colour owing to the presence of breakdown products of the poison and of the blood pigment. Neisser reported a case of fatal intoxication following inunctions of one-half the body with a 10 per cent. pyrogallic acid ointment.[1]

In clinical cases methæmoglobinæmia and methæmoglobinuria are outstanding features. The urine also contains albumin and casts.

Toxicity and Symptoms. The following cases indicate the toxicity, symptoms, etc.

A woman, *æt.* 24, married and pregnant, took by mistake for a tonic medicine a dose of "pyro" stock solution, used in developing photographs: this stock solution is made up (according to the evidence given) of—nitric acid, 20 minims; pyrogallic acid crystals, 1 ounce; water, 5½ ounces. Of this mixture she took about an ounce and a half, which would correspond to about 120 grains of pyrogallic acid.

When seen at 3 p.m. the next day, her symptoms were as follows: Body and face turned a peculiar yellowish green colour, pulse 75, temperature normal. She vomited violently, and the vomit and also her urine were of a green colour. Repeated attacks of syncope followed, with difficulty in breathing. She complained also of severe pain in the stomach and between the shoulders, and also of a "griping of the heart." She took the dose about 7.45 p.m. on Friday, September 4th, 1896. She aborted on the evening of Sunday, September 6th, and died about 7 a.m. on Tuesday, September 8th.

Post-mortem examination showed: Externally, body of a peculiar yellowish colour. Stomach corroded all over, especially at the œsophageal entrance. Perforation about one inch from œsophageal entrance. Contained about 3 drachms of a dark coffee-coloured fluid, smelling of ether. (Had taken ether to relieve pain, also hypodermic injections.)

Lungs.—Normal. *Heart.*—Normal. *Uterus.*—Slightly enlarged and right ovary congested. Contained a little congealed blood. Otherwise normal. Commencing signs of peritonitis.

Cause of death.—Perforation of the stomach, shock, syncope.

The second is thus reported[2]:—

On May 1st, at 10 a.m., I was summoned to Mrs. H. M., *æt.* 32, supposed to be dying.

Her condition on my arrival was as follows: Her face was of a dirty grey colour with the exception of her lips, cheeks, and ears, which were dark blue. She was conscious, her pupils normal, and her skin cold. The heart was acting

[1] *B.M.J.*, 1898, 2, p. 40.
[2] *B.M.J.*, 1897, 2, p. 81.

feebly, and she took no notice of anybody, but would answer questions put to her. The tongue was dark brown and dry, and she had vomited for two hours before my arrival, but that had now ceased, and the vomit had been thrown away. She complained of no pain, and exhibited no tenderness on pressure over the abdomen. I found no disease of the heart or lungs, and her mother informed me that she had menstruated a fortnight before. I then asked her relatives to leave the room, and questioned her as to whether or not she had taken poison : this she firmly denied.

The room was then searched and nothing suspicious found except a pudding basin under the bed, which appeared clean.

Large doses of ether, ammonium carbonate, and digitalis were given, mustard applied over the heart, and hot water bottles to the feet, the result being that after some time the heart acted slightly better, but there was very little improvement.

Shortly after diarrhœa set in and continued throughout the day, and the urine which was passed consisted principally of blood.

On May 2nd the diarrhœa ceased, but her appearance was unaltered : she then complained of severe headache but had no other pain and seemed in a drowsy condition, answering questions when spoken to. Later in the day her mother told her she thought she was dying, and she then confessed that she had taken poison, and that the bottle was hidden under the carpet, where it was at once found. It was an ordinary 1-ounce bottle of pyrogallic acid, used by the father for photography, practically empty, and was said to have been half full the day before. She had mixed the contents with water in the basin found under the bed, and swallowed the fluid.

On May 3rd she was decidedly worse, her face was deathlike, yet she could understand when spoken to sharply. Later in the day she became comatose, and died at 4 a.m. on May 4th, or about sixty-eight hours after the first symptoms were noticed. I tested what little remained in the bottle, and found it to be pyrogallic acid.

At the *post-mortem* examination all the viscera were intensely congested ; the kidneys were of a dark purple colour, the bladder contained blood, the heart cavities were full of clot, and the stomach and intestines showed patchy congestions.

Remarks.—The absence of gastric pain or tenderness after a large dose ($\frac{1}{2}$ ounce), the intense cardiac depression, apparently not relieved by the ordinary cardiac stimulants, the cynaosed condition with hæmaturia and the slowness of death, appear worthy of notice.

Analysis. Pyrogallic acid colours ferrous salts an intense blue, and ferric salts a brownish red ; with alkalies it becomes brown very quickly on exposure to air.

Poisoning by Thymol

Source and Mode of Occurrence. Thymol occurs in a number of vegetable oils (*e.g.*, oil of thyme). It is used as an antiseptic—*e.g.*, in gargles—and as an anthelmintic. It is much less toxic than phenol, and has very much less local action.

Toxicity and Fatal Dose. Death is said to have followed ingestion of about 90 grains, although in another case, 360 grains caused nothing more serious than diarrhœa[1]. Mild symptoms seem to be fairly common after relatively small doses[2].

Symptoms. There may be a burning sensation in the stomach, although the poison has very little corrosive action. The chief symptoms are nausea, vomiting, depression, headache, giddiness and collapse.

Analysis. Thymol, like other phenols, is separated from viscera, vomitus, etc., by steam distillation. From the distillate, it is best

[1] *J. Amer. Med. Assoc.*, 1912, 57, 1744.
[2] *Ibid.*, 1918, 70, 409.

extracted by chloroform. The chloroform solution of thymol, warmed with a little solid potassium hydroxide, develops a dark red colour. If thymol is dissolved in glacial acetic acid, and the solution is gently warmed with an equal amount of concentrated sulphuric acid, a violet-red colour is produced. Pure thymol has an odour of thyme, is insoluble in water, and melts at 51° C.

Poisoning by Caustic Potash or Soda and their Carbonates (KOH, NaOH, Na_2CO_3, K_2CO_3)

Source and Method of Occurrence. All of these substances are used extensively in the arts, and consequently prepared in enormous quantities. Cases of poisoning by them are mostly accidental. Pearlash and soap lees consist largely of these caustics.

Toxicity and Fatal Dose. As with other corrosives, concentration has much more to do with their toxicity than has the actual dose. The quantity required for a fatal dose is unknown, but **forty grains** of caustic potash has proved fatal. The metals themselves are constituent parts of the human body, and it is probable that the proportion of any of these substances that has become absorbed is practically negligible; they kill by their local action in most cases.

Duration. In acute cases the symptoms come on at once, and death takes place in a few hours. The most rapidly fatal case reported is that of a boy, who died **three hours** after swallowing three ounces of a strong solution of carbonate of potassium. In another case a child, aged three years, took a small quantity of a concentrated solution of pearlash which had deliquesced, and died in twenty-four hours. Death was caused in this instance by the inflammation induced in the larynx, causing suffocation. In this respect, the caustic alkalies may destroy life rapidly, like the mineral acids; but death may be also a slow result of these poisons. Thus a lady swallowed by mistake one ounce and a half of the common solution of potash of the shops, which contains about 5 per cent. of caustic alkali. She recovered from the first symptoms of irritation, but died seven weeks afterwards from exhaustion, becoming greatly emaciated before her death. The alkali had destroyed the lining membrane of the stomach, and had thus impaired digestion.

Such prolonged cases are by no means rare; they exhibit the symptoms of an ordinary chronic ulcer, or of stricture of the œsophagus or pylorus.

Symptoms. Caustic alkalies dissolve the mucous membranes, forming soluble alkali-proteinates. The membranes therefore swell, becoming translucent and soap-like and the sloughs are mucilaginous. The patient experiences, during the act of swallowing, an acrid caustic taste, owing to the alkaline liquid excoriating the mucous membrane. There is a sensation of burning heat in the throat, extending down the gullet to the stomach. Vomiting is not always observed; but when it does occur, the vomited matters are sometimes mixed with blood of a dark brown colour, and with detached portions of mucous membrane, this effect depending on the degree of causticity in the liquid swallowed. The surface is cold and clammy: there is purging, with severe pain in the abdomen, resembling colic, usually associated with great tenderness on local pressure, but in one recorded case this was absent. The pulse is

quick and feeble. In the course of a short time, the lips, tongue and throat become swollen, soft and red. The later or after effects are those of stricture of the œsophagus, *viz.*, pain or discomfort and difficulty in swallowing solids or even liquids, and of ulcer of the stomach, *viz.*, pain after taking food, often associated with vomiting, which may be very persistent ; there may also be stricture of the pylorus. On more than one occasion a cast of the œsophagus has been expelled by vomiting[1].

Treatment. This must be directed to neutralising the alkali by means of weak acids. Lemon juice, or vinegar, will probably be obtainable, and should be given, followed by demulcents, such as linseed tea, milk, egg-white, etc. These protein-containing fluids have a considerable buffering action towards both acids and alkalies. The stomach tube must not be used, owing to the danger of perforation. Morphia and hypodermics of ether may be given to counteract the pain and collapse. Belladonna has been recommended to decrease gastric secretion and motility. The œsophagus must be given rest before oral feeding is begun and feeding must be such as to protect rather than damage the healing mucous membranes.

Post-mortem Appearances. In recent cases there are marks of the local action of the poison on the mucous membrane of the mouth, throat, and gullet. This membrane has been found softened, detached, and inflamed in patches of a deep chocolate colour—sometimes almost black. A similar appearance has been met with in the mucous membrane of the larynx and windpipe. The stomach has had its mucous surface eroded in patches, and there has been partial inflammation. In one instance, as a result of the action of soda, the author found it puckered and blackened.

In 1891 Sir T. Stevenson gave evidence as to the death of a woman who died a few hours after drinking a 30-per cent. solution of caustic potash in mistake for iodide of potassium. The stomach was found after death almost completely dissolved in parts.

Barclay has reported a case of poisoning by potash, which furnishes a good illustration of the after-effects and appearances caused by this poison. A woman, aged forty-four, was admitted into hospital about six hours and a half after she had swallowed a quantity of American potash, probably a saturated solution of carbonate of potassium (American pearlash). She had vomited immediately after taking it. The mouth and throat were much corroded. She died from starvation on July 8th, about two months after taking the alkali. On inspection, the lower part of the gullet was found much contracted, the lining membrane entirely destroyed, and the muscular coat exposed. The external coats were much thickened. The cardiac end of the stomach, where the ulceration ceased, was considerably contracted. At the intestinal end the mucous lining presented a large and dense cicatrix, obstructing all communication with the bowels, except by an orifice no larger than a probe. The intervening portion of the stomach was healthy, as were also the large and small bowels.[2]

Chemical Analysis. Solutions of caustic potash and soda have a strongly alkaline reaction ; they are distinguished from those of their respective carbonates by giving brown precipitates with a solution of nitrate of silver. The carbonates on the other hand, yield a whitish-yellow precipitate and evolve carbon dioxide when treated with dilute

[1] Hadden, *Trans. Path. Soc.*, Lond., 1889–90, 41, p. 86; Monjour, *Gaz. Lebd. d. Sc. Med. de Bordeaux*, 1905, 26, 129.

[2] *Med. Times and Gaz.*, 1853, 2, p. 554.

hydrochloric acid in the cold. The hydroxide and salts of potassium can be distinguished from those of sodium by the following tests :

1. Moderately concentrated neutral solutions of *potassium* salts give a yellow crystalline precipitate, K_2PtCl_6, on the addition of platinic chloride. Precipitation is aided by the addition of alcohol.

2. Under the same conditions, hydrogen sodium tartrate gives a white crystalline precipitate of hydrogen potassium tartrate.

3. Hydrofluosilicic acid (silico-fluoric acid, H_2SiF_6) gives a white gelatinous precipitate, sparingly soluble in water, with *potassium* salts in concentrated solution.

4. A concentrated solution of potassium pyroantimonate (freshly prepared) gives a white precipitate with concentrated solutions of *sodium* salts.

5. Potassium nitrate crystallises in long, slender, fluted prisms ; sodium nitrate in rhombic plates.

6. Heated on a platinum wire in a colourless gas flame, potassium salts colour the flame reddish-violet or lilac ; sodium salts give a bright yellow colour.

The most important point is the *alkalinity* of the stomach contents to phenolphthalein, since such a condition has never been reported normally. If the matter tested is not alkaline, quantitative determinations of sodium and potassium are necessary.

In Liquids containing Organic Matter. Such liquids are frothy ; they possess an alkaline reaction, a peculiar alkaline odour, and are soapy to the feel. The organic liquid may be evaporated to dryness, then heated in a porcelain capsule to char the animal and vegetable matters, and the alkali will be recovered from it as carbonate by digesting the residual ash in distilled water.

Cases. In December, 1867 a case of poisoning by pearlash gave rise to a trial for manslaughter at Manchester.[1] A solution of this substance had been prepared for washing purposes. The prisoner offered some to a man, who tasted it and immediately called for water. The deceased took some, and was soon afterwards seen in the yard vomiting and in great pain. This was on May 31st ; he was admitted into a hospital, where he remained until August 2nd, suffering all the time and unable to swallow anything but thin fluids. On leaving the hospital he went home and died on September 20th, nearly four months after swallowing the alkaline liquid. He died from starvation, as a result of the stricture of the gullet. The quantity taken was unknown, but the liquid was sufficiently strong to soften and destroy the mucous membrane of the throat.

Orfila refers to two cases of poisoning by carbonate of potassium, in each of which half an ounce of this substance was taken by mistake for aperient salt. The patients, two young men, recovered from the first effects, but ultimately died : the one three months, and the other four months, after the poison had been taken. The secondary fatal effects appear to have been due to constant purging, great irritability of the stomach leading to incessant vomiting, and loss of the functions of this organ from the destruction of the lining membrane, with stricture either of the gullet or of the apertures of the stomach—either of which causes might prove fatal at almost any period. A fatal case of stricture, produced by soap lees after the lapse of two years and three months, is reported by Basham.[2]

[1] *R. v. Boothman.*
[2] *Lancet*, 1805, 1, p. 275.

Poisoning by Ammonia and its Carbonate [NH_4OH and $(NH_4)_2CO_3$]

Source and Method of Occurrence. The caustic solution of ammonia and the carbonate are common articles of commerce. As drugs they are both in common use in liniments and other preparations which are poisonous in large doses. Owing to the strong smell it is almost unknown for ammonia to be used homicidally, but there is one instance on record in which a man was tried for the murder of a child by administering to it spirits of hartshorn.[1]

Ammonia is by no means uncommon as a suicidal agent, about thirty fatal cases being recorded annually in England and Wales.

Toxicity and Fatal Dose. Ammonia is extremely volatile, and consequently easily obtains access to the air-tubes, where it sets up inflammation if the vapour be very strong or the application long continued, with symptoms that are certainly alarming and may prove fatal.

The fatal dose is quite unknown, chiefly owing to the rapid deterioration of solutions of ammonia, so that similar quantities of the liquid contain very dissimilar quantities of the real NH_4OH. In one case from one to two drachms of solution of ammonia caused death. In another instance, a man walked into a druggist's shop and asked for a small quantity of ammonia to take spots out of his clothes. The druggist poured about a teaspoonful and a half into a glass. The man suddenly swallowed it, and fell instantly to the ground. He soon afterwards died, complaining of the most excruciating pain.[2] Iliff reported the case of a little boy, aged two years, who swallowed about half an ounce of a strong solution of spirit of hartshorn, and in spite of rather severe symptoms recovered in a few days.[3].

Ammonia gas, liberated by the bursting of containers in refrigerating plants, etc., has caused serious accidents. A concentration of 0·5 per cent. in air is fatal within a few minutes, and one of 0·25 per cent. produces severe symptoms if breathed for half to one hour.

Duration. So far as onset is concerned, those symptoms which are pulmonary in origin usually occur at once. Gastric symptoms occur immediately if the stomach is empty. If the patient survives the acute symptoms, there is, as in other corrosives, the usual risk of ulceration and cicatrisation with their train of chronic symptoms. In one case a strong dose of the solution killed a man in **four minutes,** by causing suffocation (Christison). In other cases, in spite of a large dose, death has taken place slowly. Potain met with an instance in which a man swallowed upwards of three ounces of the commercial solution of ammonia, and he did not die from the effects until the eleventh day.[4] A man swallowed by mistake for a dose of cod-liver oil, a tablespoonful of solution of ammonia. Œdema of the glottis followed, and in five hours he died from suffocation.[5]

Symptoms. The strong *solution* of ammonia produces symptoms similar to those described for potash. The only difference observed is, that the sense of heat and burning pain in the throat, gullet, and

[1] *R. v. Haydon*, Somerset Spring Ass., 1845.
[2] *Jour. de Chim. Med.*, 1845, p. 531.
[3] *Lancet*, 1849, 2, p. 275.
[4] *Jour. de Chim. Méd.*, 1862, pp. 311 and 474.
[5] *Lancet*, 1870, 1, p. 467.

stomach is much greater. The suffocation, too, is usually much more marked, for serious injury to the organs of respiration is often the result of the action of this poison. In fact, in most rapidly fatal cases, death is caused by the effect of the fumes upon the glottis and air-tubes. The solution or the fumes cause a rapid inflammation with much swelling, and this naturally causes very severe asphyxia, for the fumes can easily penetrate along the bronchi, leaving a track of this swelling even to the finest tubes.

Vomiting is a prominent feature.

The damage to the eyes is serious, and may be permanent even after recovery from the other effects of the poison.

Treatment. The same as for caustic potash so far as the stomach is concerned. Tracheotomy or intubation may help the pulmonary distress, but is not very hopeful, as the trouble has probably reached below the glottis. So often is this the case that one would advise against the operation were it not that it is, humanly speaking, impossible to see a man die from asphyxia without trying to open the windpipe, however hopeless one may know it to be.

Post-mortem Appearances. The viscera usually presents strong marks of corrosion. The mucous membrane of the tongue is softened, and peeling; the lining membrane of the air-passages softened and covered with layers of false membrane, the result of inflammation, and the larger bronchial tubes deeply congested and often obstructed by casts or cylinders of this membrane. The lining membrane of the gullet is inflamed and usually softened or completely disorganised. In one case there was an aperture in the stomach in its anterior wall, about one inch and a half in diameter: the edges were soft, ragged, and blackened, presenting an appearance of solution. The contents of the stomach had escaped. On the inside, the vessels were injected with dark-coloured blood, and there were numerous small effusions of blood in various parts of the mucous membrane. The coats were thin and softened at the seat of the aperture. The blackened and congested appearance somewhat resembled that which is seen in poisoning by sulphuric or oxalic acid. The mucous matter on the coats of the stomach was feebly *acid*. No poison of any kind was found in the layer of mucus nor in the coats. There was not in any part a trace of ammonia, the poison which had caused the mischief. [The deceased had lived three days: remedies had been used, and naturally every trace of ammonia had disappeared.] In another case, the mucous membrane of the mouth and throat was destroyed. There was a bloody fluid, smelling of ammonia, in the stomach. At the lower portion, the lining membrane was corroded and the muscular coat changed into a black pulpy substance. The duodenum was also inflamed.[1] In 1871 a man was admitted into Guy's Hospital who had swallowed about a teaspoonful of, as was supposed, the stronger pharmacopœial solution of ammonia. He died suddenly not long after admission. The lips, tongue, tonsils, uvula, and pharynx were much swollen, red, glazed, with here and there flakes of white epithelium resting upon the mucous membrane. The gullet was intensely reddened throughout, and at its lower end was of a dark purple colour; but this ceased abruptly at the stomach. The epiglottis and adjacent parts were œdematous. The mucous membrane

[1] *Amer. Jour. Med., Sci.*, January 1870, p. 275.

of the windpipe and bronchi was thickened and injected. Both lungs were œdematous and gorged with blood. Both sides of the heart contained dark fluid blood. There was a circular reddened patch on the mucous membrane of the stomach, at the point on which the liquid would have first impinged ; and here the wall of the stomach was thinned.[1]

In the case of a woman who died in about three months from the time at which she had swallowed the poison, the gullet was found healthy ; the orifice, at its junction with the stomach, was slightly contracted. The intestinal orifice was contracted to the size of a crowquill, and the coats were thickened. On the posterior wall of the stomach there was a dense cicatrix of the size of half-crown, and from this point fibrous bands ramified in various directions. The duodenum and other parts of the intestinal canal were healthy.[2] This well illustrates the chronic or delayed cases.

Analysis. Solutions of the three alkalies, potash, soda, and ammonia, are differentiated from those of the *alkaline earths* by the fact that they are not precipitated by a solution of sodium carbonate. All three are strongly alkaline, but a litmus test paper, blued by a solution of ammonia, reverts to its original colour on drying. Ammonia is distinguished from potash and soda by its odour and volatility. *Carbonate of Ammonium* may be differentiated from other ammonium salts by its alkaline reaction and its odour : from pure ammonia—1, by its effervescing on being added to an acid ; 2, by its yielding an abundant white precipitate with a solution of chloride of calcium ; from the carbonates of potassium and sodium, among other properties—1, by its giving no precipitate with a solution of sulphate of magnesium ; 2, by the rich violet-blue solution which it forms when added in excess to a solution of copper sulphate ; 3, by its odour and volatility.

Cases. Tyerman attended a case in November 1858, in which a lunatic, *æt.* 62, swallowed about two fluid ounces of compound camphor liniment, which contains ammonia. The patient immediately complained of great heat in the stomach. Vomiting was induced by giving him warm water. The uvula, throat, and gullet were so intensely inflamed that he lost all power of swallowing : and the efforts to swallow liquids produced violent retching. The symptoms gradually abated, and the man recovered in four days. In this case the quantity of ammonia swallowed was small, amounting to about two and a half drachms, diluted with about six times that volume of rectified spirits. In September, 1863, Gill met with a case of the poisoning of an infant, only four and a half days old, by a small quantity of this liniment. He saw the infant about half an hour after the liquid had been taken ; it was then screaming in a suppressed manner, as if the act increased the pain ; the hands were tightly clenched ; the skin was pale and covered with a cold perspiration ; the mucous membrane of the lips was blistered, and that of the mouth and the tongue was white. A yellowish froth escaped from the mouth and nostrils ; the breathing was painful, and the pulse imperceptible. In about two hours the infant appeared better, but at intervals it suddenly started and screamed, as if from sudden pain. In six hours it continued much in the same state, and swallowing was painful. In seventeen hours the skin was moist and cool : it had had a natural motion, and had been in a drowsy state during the night. After twenty-four hours the infant was much weaker ; the limbs were cold, and the breathing was feebly performed. It became drowsy, and died thirty-two hours after taking the poison. Two cases are reported[3] in which children were poisoned by swallowing a lu-

[1] Guy's Hosp. Rep., 1872, p. 225.
[2] *Med. Times and Gaz.*, 1853, 2, p. 554.
[3] *Med. Times and Gaz.*, 1855, 1, p. 526.

ment of ammonia and oil. In one, an infant, death occurred speedily, probably from swelling and closure of the air-passages, thus leading to suffocation. In the other case, death took place on the following morning. Considering the pungent taste of ammonia, it is remarkable that an infant could have had the power of swallowing nearly two ounces of strong ammonia liniment. It had been poured down its throat by another child of five years of age. A strong solution of ammonia has been maliciously used for throwing on the person. It must be regarded as a corrosive liquid, capable of producing serious injury. Several cases of severe injury have resulted from the accidental spilling of a strong solution of ammonia on the person.

Group 2. POISONING BY THE METALLIC ELEMENTS AND BY THEIR SALTS

A study of this group will show that it has nothing to recommend it as a group except what may be termed an "index" arrangement, or a principle of origin. It must be admitted that the group is a purely artificial one; and not only so, but any serial arrangement of the metals themselves is, from a toxicological point of view, arbitrary, and one only follows the chemical grouping of the metals for convenience, though it is possible that, just as chemistry has grouped the metals by their affinities and likenesses in forming salts, so it may some day be discovered that the metals ultimately poison by these affinities for the tissues in similar groups. For instance, antimony and arsenic both show some tendency to produce fatty degeneration, and their nearest ally in this as in the chemical classification of the elements is the non-metal phosphorus.

We shall take them in the following order :—

Potassium salts.
Sodium salts.
Silver salts (the nitrate).
Barium salts.
Magnesium salts (the sulphate).
Zinc salts (the sulphate and the chloride) (Burnett's fluid).
Mercury and its salts.
Copper salts, and brass, an alloy of zinc and copper.
Gold salts, the chloride.
Thallium preparations.
Tin salts.
Lead and its salts, type metal (antimony and lead).
Arsenic and its salts. (For gaseous arsenic compounds, *vide* "Poisoning by Gases," Group 4).
Antimony and its salts.
Bismuth salts.
Osmium tetroxide.
Chromium, as chromic acid and bichromate of potassium.
Iron salts.
Nickel and cobalt.
Uranium salts and salts of other radioactive elements.

Potassium Nitrate (KNO_3), Nitre, or Saltpetre

Source and Method of Occurrence. The salt is commonly used in medicine and is an ordinary commercial substance. Cases of poisoning by it are very rare. "Warner's Safe Cure" is essentially a solution of nitrate of potassium.

Toxicity and Fatal Dose. The toxicity is very feeble. Peterson Haines and Webster [1] report the death of an adult from a dose of 8 grams (128 grains). On the other hand, doses of an ounce and more have been survived. The official dose is five to fifteen grains.

Duration. The symptoms appear within about half an hour of swallowing the substance, and cases are recorded of death in two and in three hours. Chevalier [2] records a case in which death occurred in forty-five minutes.

Symptoms. From the recorded cases these would seem to consist of pain in the stomach, soon followed by collapse (effect of absorption); vomiting and purging may occur, and if so they are rather favourable symptoms.

Treatment. Must be directed to emptying the stomach by the stomach tube or by an emetic: there is no direct antidote. Beyond this, *vide* pp. 250 *et seq.*, for general treatment.

Post-mortem Appearances. In one case the stomach was found highly inflamed, and the membrane detached in various parts. Near the pylorus the inflammation had a gangrenous character. A large quantity of bloody liquid was found in the stomach. In another case, which proved fatal in sixty hours, where an ounce and a half of nitre had been taken, a small perforation was found in the stomach. In another, on examining the body, bloody mucus was found in the stomach, the lining membrane was of a brownish-red colour, generally inflamed, and in parts detached from the coat beneath. None of the poison could be detected in the stomach; but its nature was clearly established from the analysis of a portion left in the vessel which had contained the draught.

Analysis. For the chemical properties and method of detecting the components of this salt, see pp. 329 and 300.

Cases. A case is reported by Orfila in which a lady swallowed, by mistake for another salt, an ounce of saltpetre. In a quarter of an hour there was vomiting and purging, the muscles of the face were convulsed, the pulse was weak, the respiration difficult, the limbs cold, and there was a sense of burning heat and severe pain at the pit of the stomach. She died in *three hours*. Geoghegan met with the following case:—A man took an ounce and a half of nitre by mistake for Epsom salts. Severe pain in the abdomen followed, with violent vomiting, but no purging so far as could be ascertained. He died in about *two hours* after taking the salt. Fuller had a case which proved fatal in 1863. A man swallowed an ounce of nitre, mixed with water, by mistake for Epsom salts. It produced vomiting, with severe pain, but no purging. There was coldness of the surface and lividity of the face. Death took place in three hours. On inspection the mucous membrane of the stomach was found highly inflamed, especially towards the middle of the greater curvature, where for several inches it resembled scarlet cloth. The pylorus and duodenum were of a deep crimson colour. The peritoneal surface was very vascular, especially over the stomach.

[1] "Legal Medicine and Toxicology," 1923, i., 159.
[2] "Ann. d'Hyg.," 1861, 25, 17, 400.

the vessels having a vermilion red colour, as if they had been injected. The heart and lungs were healthy ; the blood was fluid and more florid than natural. The other organs presented no unusual appearance. No analysis was made of the contents of the stomach, but that the nitre was the cause of death no doubt could be entertained, and a verdict was returned accordingly at the coroner's inquest.

In 1882 a farm labourer took an ounce of nitre in mistake for Epsom salts at 7 a.m. When seen at 1.30 p.m., he was suffering from intense pain in the stomach of a burning character, with hot distressing eructations. The pain had come on immediately after taking the salt, with profuse perspiration. He felt sick, but did not vomit till 11 a.m. Vomiting was then free, and the ejected matters were of a coffee-ground colour, and apparently contained altered blood. The pulse was 56, and full and slow ; the tongue moist, white, and tremulous. Micturition was frequent for the first three hours. Later in the day a liquid stool, of tarry appearance, was passed, and very offensive in odour. From this time he made a gradual recovery.[1] Two cases are reported of recovery after the administration of two ounces of nitre.[2]

Sulphate of Potassium, Sulphate of Potash, Sal Polychreest, or Sal de Duobus

Source and Method of Occurrence. The salt is in use in medical practice, and such cases as have occurred in England have all been accidental, but according to Mowbray,[3] sulphate of potassium is much employed in France as a popular abortive. He quotes several instances in which, in large doses, it produced severe symptoms (resembling those of irritant poisoning), and even death.

Salt cake, or nitre cake ($KHSO_4$) or ($NaHSO_4$), sold under the name of " Harpic " for cleaning waterclosets, etc., is now in common use.

Toxicity and Fatal Dose. The salt has feeble toxic properties.

The question whether it is to be regarded as an irritant poisonous salt or not was much debated among the members of the profession, in reference to a case which was tried at the Central Criminal Court in October 1843.[4]

The accused had given to the deceased, the night before her death, two ounces of sulphate of potassium, dissolved in water ; and it was alleged that a fortnight previously to this she had taken, in divided doses, as much as a quarter of a pound of the salt. The woman thought that she was pregnant, but this was disproved by an examination of the body ; and it was charged that the prisoner had given her the salt with the intention of causing a miscarriage. After the last dose, she was seized with sickness, and died within a very short time. The stomach was found empty, but highly inflamed ; and there was blood effused on the brain. One medical witness referred death to the action of this salt as an irritant poison ; the other to apoplexy, as an indirect result of the violent vomiting caused by it. The prisoner was acquitted of the charge of murder, but subsequently found guilty of administering the sulphate with intent to procure abortion.

Both of the witnesses admitted that, in small doses, the salt was innocent : but that in the dose of two ounces it would produce dangerous effects. A case, somewhat similar in its details, was the subject of a trial at the Central Criminal Court in October 1856.[5]

[1] *B.M.J.*, 1882, 1, p. 304.
[2] *B.M.J.*, 1877, 2, p. 520 ; 1882, 1, p. 500 ; also Harnack, *Vierteljahrsschr. f. ger. Med.*, 1914, 47, 257.
[3] *Med. Gaz.*, vol. 33, p. 54.
[4] *R.* v *Haynes.*
[5] *R.* v. *Galor.*

A married woman, the wife of the prisoner, in the belief that she was pregnant took a large quantity of this salt, the prisoner having purchased two ounces, and handed it to her. It was taken with the design of procuring abortion, but it caused the death of the woman with symptoms of severe irritation of the stomach and bowels. The deceased was not seen by a medical man while living, but she suffered from severe pain, vomiting, and purging; the vomited matter had a bilious colour. On inspection, the stomach and the upper portion of the small intestines were of a deep purple colour, as if from the action of some irritating substance. The stomach when opened showed marks of irritation, and its mucous coat was much congested. In this organ there was a spoonful of a thick slimy fluid, which contained a quantity of sulphate of potassium. The intestines contained twelve ounces of a thick white fluid, highly charged with mucus, and this when analysed yielded sulphate of potassium. There was no doubt that death had been caused by an overdose of this salt.

In the cases here related there is no question but that excessive doses were administered, but there is a principle with regard to saline purgatives (and indeed to other medicines) that it is as well to bear in mind, *viz.*, that if they fail for any reason to effect their ostensible purpose of purging, they are liable to be absorbed and then to cause depressant effects upon organs with which the blood brings them in contact.

In one case two drachms acted powerfully; and in another four drachms of the salt administered to a lady after her confinement, had all the effects of an irritant poison. These cases are the only instances in which it is publicly known to have proved fatal in England; and they show that substances, commonly regarded as innocent, may sometimes give rise to important questions in toxicology.

Duration. A lady, about a week after her delivery, took, by the prescription of her medical attendant, about ten drachms of this salt in divided doses, as a laxative. After the first dose, she was seized with severe pain in the stomach, nausea, vomiting, purging, and cramps in the limbs. These symptoms were aggravated after each dose: she died in **two hours.** It was supposed that some poison had been taken by mistake; but that was not the case, and the question was, whether her death was or was not caused by sulphate of potassium. On an inspection of the body, the mucous membrane of the stomach and intestines was pale, except in the valvulæ conniventes (folds), in which it was reddened. In the stomach was a large quantity of a reddish-coloured liquid, which, on analysis, was found to contain only sulphate of potassium, and no trace of any other common irritant poison. The examiners referred death to sulphate of potassium taken in an unusually large dose, whereby it had acted as an irritant poison on a person whose constitution was already much debilitated.[1]

Symptoms. Are those of an irritant, rapidly followed by the usual symptoms of depression and collapse; *vide* the narrated cases above.

Treatment. Must be symptomatic: the stomach may be emptied if thought advisable.

Post-mortem Appearances. Those common to most irritants, illustrated by the cases.

Analysis. It is said that sulphate of potassium has in some cases caused vomiting and other serious symptoms, from its containing as impurity sulphate of zinc. This, if present, would be easily discovered

[1] "Ann. d'Hyg.," April, 1842.

by the appropriate tests. A more serious impurity is arsenate of potassium. This impurity may be derived from sulphuric acid used in its manufacture. It would be well to test for arsenic any sample of sulphate which has caused great irritation.

Sulphate of potassium is easily identified. It is a dry, hard salt, soluble in water, forming a neutral solution. This solution, if sufficiently concentrated, is precipitated both by tartaric acid and by platinic chloride, whereby potassium is indicated; and the presence of sulphuric acid is detected by the action of a salt of barium (p. 295).

In Organic Liquids. The substance containing the salt may be evaporated to dryness and incinerated, when the undecomposed sulphate may be obtained by lixiviating the calcined residue with distilled water. Sulphate and potassium exist naturally in animal fluids, but only in small amounts.

Potassium Iodide (KI). See also Iodine, p. 517.

Source and Method of Occurrence. The salt is in constant use in medical practice, and cases of poisoning usually occur from misadventure. Sodium iodide is less commonly used, but can give rise to similar toxic effects.

Toxicity and Fatal Dose. There is no standard for the toxicity of KI; the salt is pre-eminently an example of that class of substances against which certain people have an idiosyncrasy, and for which a large power of tolerance may be established by gradually increasing the dose. As much as 300 grains per diem was administered to a patient for some weeks without any unpleasant symptoms; on the other hand, death is recorded[1] from as little as twenty-four grains. With such discordant evidence it is impossible to lay down any rules except that children seem to be especially prone to be affected by it. Doses of five, nine and twelve grains are recorded below as causing dangerous symptoms.

Duration. Symptoms of iodism rarely commence till the drug has been taken for some time, but exceptionally coryzal symptoms may appear very rapidly, owing to idiosyncrasy. We know that the salt is absorbed very rapidly from the stomach and intestines, and can be recognised in the saliva and urine in a few minutes.

Symptoms. In the fatal case mentioned above, a pemphigoid eruption in nose, mouth, throat and larynx occurred, with bloodstained stools.[2] In non-fatal cases the symptoms resemble an intense cold in the head as a rule, occasionally accompanied by a purpuric or acne-like eruption and sometimes associated with intense œdema of the mucous membranes.

A gentleman was ordered by his physician to take three grains of potassium iodide in peppermint-water three times a day. After the third dose he felt unwell, and an hour after the fourth dose he was attacked with a violent shivering fit, followed by a headache, hot skin, intense thirst, quick full pulse, with vomiting and purging. These symptoms were succeeded by great prostration. In spite of treatment, the purging lasted several days. The effects of the medicine in this case were so violent, although only *twelve grains* had been taken, that there is little doubt, if the patient had taken another dose, he would have

[1] *New York Jour. of Med.*, 1850.
[2] Mann, "For. Med."

died.[1] In October 1841, a case was reported by Erichsen to the University College Medical Society, in which alarming symptoms resulted from a dose of only *five grains* of iodide of potassium. There was great difficulty of breathing, discharge from the eyes and nostrils, inflamed conjunctivæ, and most of the violent symptoms of catarrh. The iodide was discontinued, and the patient recovered. Lawrie found that seven and a half grains of the iodide, in three doses, produced in an adult dryness and irritation of the throat, great difficulty in breathing, and other serious symptoms. In another instance, thirty grains, in divided doses, caused severe headache and secretion of tears. In two instances, wherein he had prescribed it medicinally in small doses, it was, in his opinion, the cause of death.[2]

Treatment. The only treatment is to stop the administration of the drug or combine it with a little arsenic. Doubling the dose, which used to be recommended, is a very unsafe procedure ; any other treatment must be purely symptomatic.

Post-mortem Appearances. These are not characteristic, but frequently acute œdema of the glottis, eruptions in the mouth and fauces, œdema of the lungs, and occasionally spots of hæmorrhage in the mucous membranes may be observed.

Analysis. Iodides are decomposed by concentrated sulphuric acid, or by oxidising agents, such as nitric acid, chlorine water, etc., with liberation of iodine. The iodine can be detected by (*a*) giving a deep blue colour with starch (sulphuric acid destroys the starch ; nitric acid or chlorine water are therefore better agents for liberating the iodine), (*b*) forming a violet solution with chloroform. The salt gives a violet colour to flame, indicative of potassium, and yields purple vapour of iodine when heated with concentrated sulphuric acid and manganese dioxide. Precipitation of the iodides of the heavy metals, especially of silver (yellow, insoluble in nitric acid or ammonia), mercury (red) and of lead (yellow, soluble in hot water, and crystallising from solution in shining plates), may be used as further confirmatory tests.

Potassium Bromide (KBr). See also Bromine, p. 516.

Source and Method of Occurrence. Like the iodide, this salt is in constant use in medicine, and cases occur from time to time in which its use gives rise to general symptoms of bromism, and rashes on the skin. Occasionally death may ensue from œdema or exhaustion, but we know of no recent cases in which there has been a fatal result except from extremely large doses.

Toxicity and Fatal Dose. Precisely the same general remarks may be made on this salt as on the iodide, and in all probability there is the same large variation in dosage. Many cases are known in which as much as 280 grains a day has been taken with good results, whereas as little as ten grains given over a period of twelve hours has produced very troublesome effects. Children seem to be very prone to show toxic effects from small doses.

[1] *Med. Gaz.*, September 3rd, 1841.
[2] *Med. Gaz.*, 26, p. 588.

Duration. In two fatal cases sleep was procured after an ounce and a half had been given over a period of twelve hours, and the sleep terminated in death in the one case after eighteen hours, and in the other after about six hours. In the cases in which the rash causes death there is commonly a duration of the case of from five or six days to as long as three weeks, either to death or recovery.

Leschke quotes a case reported by Vilén, in which death from bilateral pneumonia occurred six days after the enormous dose of 1,600 grains (100 grams) of sodium bromide within thirty-six hours.

Symptoms. Commonly, if bromide cannot be tolerated, there is a certain amount of nausea, vomiting and colic, but it is not possible to say much more of immediate symptoms. If a rash occurs it generally appears within a few days of the commencement of taking the drug, though exceptionally after a long period of tolerance a rash may appear. A sort of mental hebetude is common in those who are taking the drug in large doses, though it may be difficult to separate this from a post-epileptic condition not due to the drug. In the fatal cases described below, the quickening of the respiration led to a suspicion of pneumonia, though none was present; this was preceded by progressive drowsiness culminating in a deep sleep of an apparently normal character, except that the patient could not be roused.

Treatment. Little can be said upon this from a toxicological point of view. The salt is very rapidly absorbed from the stomach, and may, like the iodide, be distinguished in the saliva and urine within a few minutes of its entrance into the stomach, so that emetics and purgatives are useless. Alcohol, ammonia, hot coffee, and other rapidly acting stimulants may be tried, as also hypodermics of strychnine (one-thirtieth of a grain repeated in an hour). If the patient lives five or six hours there is a hope of recovery if the kidneys are sound.

In ordinary medicinal cases the drug must of course be at once stopped. For further treatment *vide* works on medicine.

Post-mortem Appearances. These are not in any way characteristic.

Analysis. The contents of the stomach or the blood, etc., may be filtered or dialysed and the ordinary tests applied. The following tests are very sensitive and are not given by chlorides (which, of course, are always present in biological material).

1. Put a drop or two of the solution under test into a small test-tube of which the neck has been drawn out to a tube of about 2 mm. diameter; add a drop of acetic acid and a very little lead dioxide. Over the open end of the tube place a piece of filter paper which has been soaked in a dilute alcoholic solution of fluorescein and dried. Warm the test-tube over a small flame. Bromides liberate bromine which is evolved and absorbed by the fluorescein to form the red dye, eosin. Iodides act similarly producing red erythrosin.

2. Invert a microscope slide carrying a drop of aqueous m-phenylene-diamine solution over a small crucible containing a drop of the test solution, a drop of dilute acetic acid and a little lead dioxide. Warm the crucible gently. If bromine is evolved it produces a film of 2, 4, 6-tribromo-m-phenylene-diamine over the drop of reagent and microscopic examination shows this to consist of masses of short needles. The

precipitate is readily soluble in alcohol. Iodides do not interfere, nor do nitrites, nitrates, sulphides, sulphites, chlorides, cyanides, and ammonia.

Cases. A medical man, aged about forty, became addicted to the morphia habit, and to obtain a cure submitted himself to the bromide treatment, and took a drachm every hour for eight hours: he then fell asleep with very rapid breathing, and died in about six hours. A woman, aged about 45, a chronic alcoholic, went through precisely the same experience, but lived about eighteen hours after taking one ounce and a half of the drug.

In the *Lancet* for April 4th, 1896, p. 939, is a report of an inquest held in Essex on a man who probably died from overdoses of bromide of potassium. Suffering from neuralgia, he had been in the habit of taking this salt in doses of from two to three drachms. The drug does not appear to have produced any of the usual symptoms of bromism, but may have caused the palpitation of the heart of which the deceased had frequently complained. During the night of March 23rd he felt very cold, gave two deep gasps, and almost immediately expired. The medical practitioner who was sent for stated at the inquest that he found the deceased quite dead, his mouth wide open, the eyes half closed, and the pupils somewhat dilated. A bottle containing the drug was on the table, and examination showed that it was bromide of potassium. The witness attributed death to failure of the heart's action caused by taking the bromide.

On the above case the *Lancet* remarks :—

"It proves in a marked manner the danger of taking any drug, however harmless it may be reputed to be, in large and repeated doses without the advice of a medical man. No doubt in this instance a medical attendant would have recognised the depressant action the salt was exerting upon the heart, and would have discontinued its use. As bromide is constantly administered in large and repeated doses, the action of its basic constituent should always be borne in mind, and if signs of its depressant effect are observed its use should be abandoned, the bromide of some other base being selected if in other respects the action is beneficial. Many secret remedies for 'fits' contain this drug in large quantities, and it is evidently desirable that the public should be warned that their use is not unattended with danger."

Chlorate of Potash ($KClO_3$)

Source and Method of Occurrence. This drug is freely used in medicine, and accidental cases occur from overdosage. Its use is decreasing, however, since other mouth and throat disinfectants have gained popularity.

Toxicity and Fatal Dose. The drug is not commonly regarded as a poison; the official dose is five to ten grains. Idiosyncrasy seems to have some influence, but carelessness in its administration is responsible for most cases. The smallest recorded fatal dose seems to be the following, by Brouardel[1]: A solution containing two drachms of the salt was given in divided doses every ten minutes to two children, the administration extending over a period of about three hours. The symptoms were those of gastro-intestinal irritation, with blueness of the skin, and collapse. Death took place within a short time.

McShane[2] reports a case in which a girl aged eleven years took 200 grains in two days and died on the sixth day.

[1] *Jour. de Med. et de Chir.*, December 1881.
[2] *Jour. Amer. Med. Ass.*, 1894.

Duration. Death does not take place very rapidly, that is, not within several hours, and usually several days elapse before death occurs.

Symptoms. This substance causes destruction of the red blood corpuscles and converts hæmoglobin into methæmoglobin, which is of no value as a respiratory pigment. Cushny has shown that the conversion can take place in four hours. The kidneys are usually affected moderately early, and the urine becomes scanty, dark in colour, and contains albumen, casts, and broken-down red corpuscles.

The symptoms consist of nausea and vomiting from the salt action, dyspnœa, gradually increasing cyanosis, followed by respiratory and cardiac failure. In addition there is pain in the loins from the implication of the kidneys, and urinary symptoms as mentioned above. Shortly after ingestion of the poison, the skin, especially of the face and extremities, acquires a characteristic bluish-grey colour. Icterus may appear later as a result of the hæmoglobin destruction. There is no specific action on the central nervous system.

A man, æt 18, took about an ounce of this salt in water in two doses at intervals of half an hour. In a short time he became faint and thirsty, and in two hours he vomited. There was pain in the region of the stomach, headache, constipation, and anorexia. The skin was pale, and the ears and extremities blue. Rigors and slight jaundice appeared. The pulse was good and regular. Tenderness over the regions of the stomach and kidneys supervened. The urine was very scanty, contained albumen and disorganised blood corpuscles, and eventually was suppressed ; and death occurred on the sixth day. After death fluid was found in the pleural and pericardial cavities, and there was œdema of the lungs. The whole alimentary canal was inflamed and eroded in patches. The kidneys, liver, and spleen were enlarged.[1]

Treatment. The stomach should be washed out with repeated quantities of warm water. The heart may require support with digitalis. Inhalation of oxygen and artificial respiration must be persevered with, and the kidneys attended to. Transfusion of blood may be of value, as in the following case of Becart's.[2]

A woman, aged fifty-five, accidentally took 40 grams of potassium chlorate in mistake for sodium sulphate. The following morning she was awakened by severe abdominal pain ; she vomited everything she took, and had suppression of urine. Numerous wet cups were applied to the lumbar region and gave issue to a thick brownish-black viscid blood which coagulated at once. Bécart, who was called to see the patient the next day, transfused 200 c.c. of whole blood, and a few hours later she evacuated a few drops of black urine and spontaneously passed a motion of the same colour. The following day she passed 38 grams of urine which was less dark in colour. A second transfusion was performed three days after the first, and eight days after the accident the total amount of urine in the twenty-four hours was 165 grams. Improvement was gradual, and eventually complete recovery took place. Bécart adds that the case is of interest, first, in view of the rarity of recovery after so large a dose of potassium chlorate, and, secondly, on account of the rapid destruction of the red corpuscles. Examination of the blood twenty-four hours after the drug had been taken showed that the red cells had fallen to 2,500,000.[3]

Post-mortem Appearances. The colour of the blood and the lesions in the kidney are the chief evidences of poisoning. The blood is a brown colour, and on microscopic examination the corpuscles are seen

[1] Landerer, in *Deutsch., Arch. f. Klin. Med.*, 1890, 47, p. 103.
[2] *Bull. Soc. de Ther.*, November 11th, 1925, p. 254.
[3] *B.M.J.*, Epitome, February 13th, 1926.

to be pale and shrunken, with granulated pigment in the stroma. Spectroscopic examination shows the absorption bands of methæmoglobin. The kidney is brownish-red, especially in the pyramids. Microscopically the tubules are filled with brown granular masses, the epithelium swollen and granular. The tubules may be so completely obstructed as to explain the anuria which has been observed (*vide* also case above).

Analysis. Potassium chlorate may be separated from organic matter (along with other salts) by dialysis. After concentration of the dialysate, a number of tests may be applied, most of them depending on the fact that chlorates are powerful oxidising agents.

(1) Add a few drops of a solution of potassium iodide and starch. Acidify with acetic acid. In the presence of potassium chlorate, iodine will be liberated and give an intense blue colour with the starch.

(2) Colour the solution with indigo sulphate, acidify with sulphuric acid, and add sulphurous acid. In presence of chlorate the blue colour will be discharged.

Case. The following occurred a few years ago at Morecambe :—An inquest was held at Morecambe on the body of Eric Spencer, *æt.* 2. The uncle of the child deposed that it was very fond of sweets, and to cure it of the taste he purchased some chlorate of potash tablets on Saturday afternoon while on a holiday at Morecambe, giving him about six, and two hours afterwards a similar number. Witness had occasionally used the tabloids, having himself taken a dozen to twenty in a day. On Saturday evening the child became ill. Dr. Benson stated that the child died from poisoning. It had been a great error of judgment to give the child so many tabloids, as ten grains would be fatal for a child of two years, and each tabloid should contain from four to five grains of chlorate of potash. It acted as a gastro-intestinal irritant, and an overdose was calculated to produce death in two hours. The coroner, quoting from an authority, said that the smallest fatal dose recorded was forty to forty-five grains given to a child of three. He hoped the case would act as a warning to people. The jury returned a verdict of " Accidental death from poison, unconsciously administered."

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Potassium Permanganate ($KMnO_4$)

Source and Method of Occurrence. The drug is official, and its dose is given as one to three grains. It occurs in small crystalline masses of a dark reddish-purple colour. It is soluble to the extent of 1 in 20 of water, forming a magenta-red solution. " Condy's Fluid " is a solution of the sodium salt. $KMnO_4$ is usually considered a safe disinfectant, but cases of poisoning by it are on record.

In the solid state or in strong solution it has an irritating effect on the gastric mucosa on which it exerts its main effects. After absorption it has a toxic action which is probably due more to the potassium than to the manganese.

Toxicity and Fatal Dose. As above stated, the toxicity of this substance is not great and it is now used freely as an antidote to other poisons. In the case recorded below a "handful" of the crystals proved fatal.

Duration. In the case recorded on p. 344 death ensued thirty-five minutes after taking the poison. Adler reported a case [1] in which death occurred on the fourth day after a dose of 10 grams of solid potassium permanganate.

Symptoms. There are symptoms of gastric irritation, namely pain and vomiting with signs of kidney irritation. The following case is reported :— [2]

"A married woman, aged thirty-six years, was found to be suffering from almost continuous vomiting, great pain and tenderness on pressure over the abdomen, excessive thirst, congestion of the fauces, and slight difficulty in swallowing; temperature 100° F., the tongue moist and pale, and pulse 120 per minute. The urine was scanty and high-coloured; the bowels were regular. There was slight tympanitic distension, and the legs were drawn up. The weight of the bedclothes caused pain over the abdomen. She had been in good health up to the previous day, when she was attacked with vomiting and pains in the stomach, which kept increasing. She had been taking pills three or four times daily with the object of bringing on her menses, which had not appeared for two months, and said that the pills, when broken and put in water before swallowing, turned the water the colour of 'Condy's Fluid.' Each pill contained about two grains of permanganate of potassium, and the total amount of permanganate taken by the patient during the period of four days was about twenty-two grains. Complete recovery occurred in a week."

Treatment. The stomach should be washed out, raw eggs or charcoal given to absorb the poison, after which it is again evacuated, and general measures adopted to combat shock and depression (*vide* pp. 250 *et seq.*).

Post-mortem Appearances. In the case quoted below the mucous membrane of mouth, tongue, fauces, and gullet was destroyed and charred; the non-destroyed parts were intensely congested. The larynx and trachea were inflamed, and œdema of glottis was present. The stomach was filled with dark, grumous matter, and the greater part of its mucous membrane was destroyed and blackened; similar changes were found in the duodenum; intestines otherwise healthy; other organs in a natural condition except for old alcoholic changes.[3]

It must not be assumed that these appearances nor anything like them will always be found, as no doubt the drug could kill by its effects after absorption in doses much less than "a handful." The above merely show that it can be a very severe irritant, nearly a corrosive.

Other observers describe marked kidney changes—swollen glomeruli, swollen and necrotic epithelium of the convoluted tubules, fatty degeneration especially near the main ducts.

Analysis. When unaltered, the colour of a solution of permanganate is very distinctive; the colour is destroyed, if the solution is acidified with sulphuric acid, by a variety of substances—hydrogen peroxide, ferrous sulphate, oxalic acid (in hot solution), etc. In addition the ordinary tests for manganese may be applied. In contact with organic matter, potassium permanganate is slowly reduced, and brown manganous

[1] *Med. Klinik.*, Berlin, 33 : 1914 (August 16th).
[2] *Lancet*, 1899, 2, p. 1467.
[3] *Lancet*, 1899, 2, p. 411.

oxide is produced. This substance is soluble in dilute sulphuric acid (more easily on heating) and the solution gives the tests for manganese. (Organic matter may be removed by dialysis or, sometimes, filtration.)

Case. M. C., æt. 47, a heavy drinker, was drunk on February 26th, 1899, and took in some beer a handful of the crystals. On arrival at St. Thomas's Hospital the lips and mouth were darkly stained; shock was marked, she was pale, the skin was very dry, pulse rapid but moderately strong. Slight signs of dyspnœa soon came on; two or three minutes after admission the pulse could not be felt, and respiration soon ceased.

Potassium Chromate and Bichromate

See section on poisoning by chromium (p. 462).

Alum [$Al_2(SO_4)_3K_2SO_4 + 24H_2O$]

The double sulphate of aluminium and potash or aluminium and ammonium, known as alum, is in common use as an astringent, but it acts as an irritant poison when given in large doses. Tardieu, who gives the details of two cases of fatal poisoning by alum,[1] is of opinion that from half an ounce to an ounce of the salt is a fatal dose for an adult. In 1888 Bull communicated to Sir Thomas Stevenson a case in which a diptheritic child, *æt.* 3 years, died from the effects of a teaspoonful of alum given in syrup as an emetic. The child did not vomit, and died shortly after. The mucous membrane of the stomach was red and velvety, as if from the effects of a powerful irritant.

Analysis. The material to be examined is incinerated in a platinum dish, and the residue dissolved in hot hydrochloric acid and filtered.

Aluminium salts give no precipitate with hydrogen sulphide; with sodium hydroxide they give a white precipitate which dissolves in excess of the reagent.

Atack's Test. 1 c.c. of a 0·1 per cent. solution of alizarin monosulphonic acid is added to about 5 c.c. of the neutral or acid solution and ammonia added until alkaline, as shown by the purple colour. Boil, cool and acidify. A red colour or precipitate indicates aluminium.

Sodium Borate, or Borax, and Boracic Acid

Borax, which is the sodium salt of boracic acid, and boracic acid have been used largely as mild antiseptics and also as food preservatives. The departmental committee on the use of preservatives (Report, 1924) stated that a single dose of boracic acid took about five days to be excreted, so that if food preserved with boracic acid was constantly taken, the body would never be free from it. Although boracic acid is generally looked upon as a harmless mild antiseptic and is used freely for a number of purposes without ill effects in ordinary circumstances, there appears good reason to suppose that the continued use in small doses is likely to have a deleterious effect on health and that it is capable of causing death when it is absorbed in large doses. It causes loss of appetite, gastro-enteritis, nervous prostration, and muscular weakness. It is also liable to cause various skin rashes. Its use as a food preservative has now been prohibited.

[1] "L'Empoisonnement," p. 218.

Fatal cases have been reported after irrigation of the bladder and abscess cavities with a saturated solution and after its ingestion as an abortifacient.

The accidental administration of a solution of boracic acid to a number of babies in a Chicago hospital resulted in the death of six of them[1] the total quantity taken per child being approximately 3–6 grammes. A case of fatal poisoning in a woman who ingested half an ounce as an abortifacient is reported.[2]

Rinehart[3] records two rare cases of the symptoms of poisoning with boracic acid, and points out the importance of the subject in view of the general belief in its supposed innocuousness. Case 1.—A man, aged thirty-eight years, had posterior urethritis treated with weak $AgNO_3$ solution locally, and five-grain doses of boracic acid by the mouth every four hours. Two days later there followed extreme weakness, and an erythematous rash beset with papules and vesicles developed on the back of the hands and between the fingers. Pulse weak, but not accelerated. The symptoms subsided slowly upon withdrawal of the boracic acid, and reappeared on resuming the drug. The case would probably have ended fatally if the cause of this alarming collapse had escaped detection. Case 2.—Man, aged fifty years, had a suprapubic lithotomy performed on him, and the bladder washed out daily with saturated solution of boracic acid, and five grains of the drug given by the mouth every four hours. Ten days after the operation there appeared an erythematous rash about the wound and spreading over the hypogastrium. Scales and crusts formed on the rash, and the skin thus affected became thickened and infiltrated as in eczema. On discontinuing the boracic acid the eruption slowly disappeared, and on resuming the drug it reappeared in two days. The drug was slowly eliminated from the system, and hence the rash could be made to reappear with ease. Albuminuria and weakness became prominent symptoms during the appearance of the rash in this patient ; the pulse was feeble, nausea was present, and at the height of the eruption the temperature rose one or two degrees above the normal.

The original article states that in the first case also the bladder was washed out periodically with a concentrated solution of boracic acid. Washing out body cavities and the bladder with very large doses of boracic acid in cases like the above, that is, where the epithelium is not in a normal condition, may entail serious consequences. A concentrated solution of boracic acid is a 3 per cent. solution, and one or two litres of it are often employed, so that about sixty grams, that is, about two ounces, of boracic acid pass through the bladder, which thus has occasion to absorb a far larger amount of boracic acid than is ever given by the mouth.

Dr. H. Sinigar reported the following case (*Lancet*, August 4th, 1917, p. 162) :—

"I was called at 9.30 a.m. to see a woman of seventy years who, at 4 a.m. on the same day, had taken a teaspoonful of boric acid in mistake for Epsom salts. I found her with a flushed face, but with no other symptoms than the mental distress produced by her mistake. She had already vomited, and I therefore did not consider it necessary to do more than administer a mixture containing carbonate of magnesium and bicarbonate of soda. During the afternoon she vomited several times, and there was some diarrhœa. A fairly comfortable night and day followed. On the next evening great thirst supervened, but she appeared to settle down into a normal sleep. She died in her sleep forty-six hours after taking the acid.

"On *post-mortem* examination there was found on the posterior surface of the stomach near the middle of the greater curvature an area about 2 inches square showing numerous circular pock-like erosions of the mucous membrane. The whole thickness of the mucous coat was eroded, the sero-muscular coat being exposed in the floor of each erosion. The right auricle contained a large, firm, *ante-mortem* clot, the cusps of the mitral valve were thickened, and there was a small granulation on each cusp."

[1] McNally, W. D., *Journ. A. M. A.*, 90 : 382 (1928).
[2] Schwyzer, *Journ. A. M. A.*, 76 : 385 (1921).
[3] *Therap. Gaz.*, October 15th, 1901 [*B.M.J.*, Epitome, 2 : 401 (1901)].

In a case cited by Potter[1] the patient took what he thought was a saline cathartic. Within fifteen minutes he was seized with violent epigastric cramps, accompanied by attacks of retching and vomiting, coming on at from five- to ten-minute intervals. At five-minute intervals he would cry out that he was strangling, or choking. The lips were slightly cyanotic. At intervals of a few minutes he cried out with pain in the epigastrium, and then immediately after he would grasp his throat and complain of difficulty in swallowing, of choking and strangling. Notwithstanding active hypodermic stimulation ($\frac{1}{20}$ grain of strychnine and 2 c.c. of camphor in oil), he died in one of these paroxysms soon afterward. There was no lead line on the gums or other symptoms present or past to suggest lead colic. Chemical analysis of the contents of the stomach demonstrated the presence of 1½ oz. of borax. At the coroner's inquest it was shown that the deceased's mother-in-law, about eighty years of age, had filled the empty saline bottle with borax without informing the family or changing the label, and that it had been placed on the medicine shelf by another member of the family who thought it was a bottle of saline laxative.

A case of boric acid poisoning occurred some time ago in a Hungarian village. The patient, a man aged twenty-three, suffered from bilateral inguinal adenitis, which had been incised. The patient packed the cavities of his buboes with boric acid powder. He used for this purpose about 150 grains. Soon afterwards the man developed signs of boric acid poisoning, profuse vomiting, a papular rash over the face, neck, and chest, and a weak irregular pulse. The onset was rapid, and the fatal issue came on within four days. There was also delirium and slight rise of temperature. *Post-mortem* examination showed congestion of the liver, spleen, and gastro-intestinal tract, and cloudy swelling and granular degeneration of the liver and kidney cells. There were also subpericardial hæmorrhages, together with discoloration and maceration of the tissues about the cavity in which the boric acid was placed. These findings limit the cause of death to a toxæmia resulting either from the abscess in the groin or from the medicinal agent employed, as other recognisable causes were not present. Its resemblance to other cases previously reported (the last one described by Dr. C. I. Best in America), together with the demonstrated over-use of the drug, was sufficient for the diagnosis. The clinical features and the lack of success in cultivating pathogenic bacteria from the blood eliminate septicæmia, which is the condition most likely to be simulated in such a case.

The official dose of borax is five to fifteen grains, and the evidence seems distinctly to point to this as safe. Such doses have no abortifacient effect, although such has been suggested.

The *treatment* of boric acid poisoning is mainly symptomatic. The stomach should be washed with lime water, or a solution of calcium chloride, 30 grains to the ounce. The intravenous administration of calcium gluconate has been recommended, but such treatment should be adopted with caution.

Analysis. The material is made strongly alkaline with sodium hydroxide and (if liquid) evaporated to dryness or (if solid) repeatedly extracted with hot water and the extracts evaporated. The residue is incinerated and the ash dissolved in sulphuric acid.

Tests. 1. Acidify the solution in a basin with sulphuric acid, float alcohol on the top and set on fire. The flame is coloured green. 2. A drop of the suspected liquid is dried on a water-bath with two drops of hydrochloric acid and two drops of a saturated solution of turmeric. To the dry residue is added a drop of strong ammonia. A blue colour, changing to green, indicates the presence of a borate.

Poisoning by Zinc Salts

Source and Method of Occurrence. The sulphate is employed to some extent in medicine, mainly as an astringent and as an emetic. In larger

[1] *Journ. Amer. Med. Assoc.*, February 5th, 1921.

doses it causes acute gastric irritation. The emetic dose is 10-30 grains so that it cannot be considered a dangerous poison.

Tardieu and Roussin have published a case of criminal poisoning by sulphate of zinc administered in soup. A woman, *æt.* 60, died in three days with the usual symptoms of irritant poisoning (gastro-enteritis). Zinc was detected in the coats of the stomach and intestines, as well as in the spleen and liver.[1]

The chloride is strongly caustic and causes severe gastric corrosion on ingestion. It occurs as a disinfectant under the name of "Burnett's Fluid," which is a highly concentrated solution of the chloride, containing about 220 grains of the salt per fluid ounce. It is used as a flux in soldering. It has been taken by accident in several cases, and in one instance was supposed to have been criminally administered as a poison.

The oxide is largely used in medicine as an ingredient in ointments, dusting powders and lotions. Leschke[2] quotes a case of non-fatal though severe poisoning from the ingestion of 10 grams of zinc oxide triturated in lemon juice.

For an account of wholesale poisoning by drinking water impregnated with a zinc salt, probably the carbonate, *vide B.M.J.*, 1901, 2, p. 615.

A case is reported[3] in which a girl, *æt.* 13, died from zinc poisoning in which the metal was taken in food prepared in cooking pans coated with an alloy containing zinc, and severe cases have occurred from the contamination of food cooked in galvanised iron pans.[4]

Toxicity and Fatal Dose. Neither the sulphate nor the oxide of zinc can be regarded as powerful irritants, although they are usually described as poisons.

In one case a lady recovered after taking sixty-seven grains of zinc sulphate.[5] In another, a man, *æt.* 20, recovered in a few days after taking an ounce of sulphate by mistake for Epsom salts. There was early vomiting and purging of a most violent kind, and great prostration.

In cases of epilepsy, sulphate of zinc used to be given in doses of forty grains three times a day, first commencing with small doses. As a rule no ill effects followed, and none of the usual symptoms of irritation were observed With respect to *oxide of zinc*, an epileptic took as much as one pound in seven months, the largest quantity taken in one day being seventy grains. Although he did not suffer from the remedy, the disease was not cured.[6]

The chloride, or at least "Burnett's Fluid," is much more toxic, but the exact doses of the chloride are not available. Luff says six grains have proved fatal: there can be no doubt but that the free acid present is an important factor.

R. Englemann,[7] records the case of a patient aged forty-five who douched herself thrice daily with 1 drachm of zinc chloride solution (50 per cent.) to 1 litre of water. After the second injection made during the menstrual period she was suddenly seized with acute abdominal pain, nausea and vomiting; the abdomen was swollen and

[1] "Ann. d'Hyg.," 1871.
[2] "Clinical Toxicology," 1933, p. 83.
[3] *Lancet*, 1909, 1, p. 1137.
[4] *B.M.J.*, 1: 201 (1923).
[5] *Lancet*, 1856, 1, p. 540.
[6] *Ibid.*, 1862, 1, p. 224.
[7] *Deuts. Med. Woch.*, 48: 488 (1922).

tender, the pulse was small, and the urine showed the presence of albumen and casts. Convalescence occupied six weeks. The conclusion is drawn that patients douching themselves on account of gonorrhœa or other maladies should be warned to suspend during menstruation the injections of zinc chloride, silver nitrate, or mercury oxycyanide.[1]

The dangers of *zinc stearate* dusting powders are pointed out by the *Jour. Amer. Med. Assoc.*, July 12th, 1924, in which records are given of many cases of acute poisoning and broncho-pneumonia from inhalation of the dusting powder.

McCord[2] reports cases of severe dermatitis from zinc chloride used in the wood preserving industry.

Duration. In an acute case, the shortest recorded time till death is **two hours** (*vide* below). If death does not take place early, recovery is usually rapid except in the case of the chloride, which undoubtedly produces gross organic change in the mucous membrane of the stomach as well as a simple acute gastritis. The latter may pass off in a fortnight or so, but the former may lead to any or all of the ordinary results of a gastric ulcer, *i.e.*, hæmorrhage, perforation or scarring, with blockage of the exit from the stomach, and to these no approximate limit can be assigned.

In one case, about two ounces of a solution containing only twelve grains of the chloride were swallowed. The patient immediately felt pain and nausea ; vomiting followed, and she recovered, but suffered from some indisposition for three weeks. In a second case a wineglassful, equivalent to at least two hundred grains of solid chloride, was swallowed. The man instantly experienced a burning pain in the gullet, burning and griping pain in the stomach, great nausea and coldness. Vomiting came on in two minutes ; the legs were drawn up to the body ; there was cold perspiration, with other signs of collapse. He recovered in sixteen days.[3] In 1863 a girl took a wineglassful of " Burnett's Fluid," and died in less than two hours.

Symptoms. The symptoms produced by an overdose of sulphate of zinc, which is a powerful emetic, are pain in the abdomen and violent vomiting, coming on almost immediately, followed by severe purging. As with other metallic emetics and purgatives if the immediate alimentary effect is not produced, there is danger that the salt will be absorbed and produce deleterious effects on the nervous system and on the heart muscle.

A concentrated solution of the chloride has a strong corrosive action, destroying the membrane of the mouth, throat, gullet, and stomach. Vomiting is severe, there is purging with tenesmus and blood-stools and great prostration. In cases which do not die there may be remission of symptoms and recurrence similar to those in arsenic poisoning.

Treatment. Sodium bicarbonate dissolved in warm water may be given by the mouth and unless it is vomited the stomach should be washed out with this solution. White of eggs and milk may be administered and medicinal charcoal may be used to absorb any of the drug not otherwise eliminated. Vomiting occurs from the poison so freely that there is usually no necessity to empty the stomach. General measures to counteract shock and collapse must be adopted (*vide* p. 250).

[1] *B.M.J.*, Epitome, December 22nd, 1922.
[2] *Jour. Amer. Med. Assoc.*, February 12th, 1921.
[3] *Edin. Med. and Surg. J.*, 1848, p. 335.

Post-mortem Appearances. These are not characteristic of zinc, but are similar to those of other irritants in the case of the sulphate and to those of other corrosives in the case of the chloride.

In the case of an infant, aged fifteen months, who died from the effects of this poison, the lining membrane of the mouth and throat was white and opaque. The stomach was hard and leathery, containing a liquid like curds and whey. Its inner surface was corrugated, opaque, and tinged a dark leather hue. The lungs and kidneys were congested.

In another case, with the exception of the cardiac orifice, which was healthy, the whole of the mucous membrane of the stomach was of a deep red colour, thickened and softened. This redness was uniformly intense, as far as that part of the fundus which lay in contact with the spleen; here the redness was deeper than elsewhere, and there was an ulcer of the size of a penny-piece, which had penetrated the coats of the stomach, but had been prevented from perforation, partly by the spleen, which formed the floor of the ulcer, partly by adhesions which passed from the spleen to the stomach. There was also congestion along the whole length of the intestinal canal.

Analysis. The material to be examined is digested for some hours on the water bath with dilute (10 per cent.) acetic acid and filtered, or is incinerated at a low temperature and the ash dissolved in sulphuric acid. A solution of ammonium sulphide is then added and the white precipitate (which is formed if zinc is present) is filtered off, washed, and dissolved in boiling hydrochloric acid.

Tests. 1. Ammonium or sodium hydroxide gives a white precipitate soluble in excess of the alkali.

2. Potassium ferrocyanide gives a white precipitate.

3. Sulphuretted hydrogen or ammonium sulphide gives a white precipitate from neutral or alkaline solutions. [The latter reagent, in excess, gives a precipitate even from acid solutions, but confusion may result from the precipitation of sulphur.]

4. Magnesium ribbon, placed in the neutral solution, causes precipitation of metallic zinc as a grey powder.

5. Evaporate the solution to dryness and heat a little of the solid residue on platinum foil in the oxidising flame. A zinc residue will be yellow when hot and white when cold. Moisten with a drop of cobalt nitrate and heat again; a zinc residue will turn green.

Zinc sulphate closely resembles Epsom salts and oxalic acid, forming white prismatic crystals. Epsom salt (magnesium sulphate) does not respond to any of the tests given above; oxalic acid, besides giving negative results with these tests, disappears when heated.

In dealing with the pure material, the actual salt of zinc can be detected by applying the usual tests for acid radicles (barium nitrate for sulphate; silver nitrate for chloride, etc.). In dealing with stomach contents, tissues, urine, etc., this is usually not possible, though the presence of large amounts of sulphate in stomach contents or vomitus is suggestive of ingestion of that salt. The chloride is sometimes used for the preservation of the dead body. This might account for the occasional presence of traces of zinc.

Cases. The following is reported by Dr. Mackintosh.[1] The packet contained one ounce of the sulphate, but it is possible that the victim swallowed two ounces:—

I was called to see A. C., a widow, aged fifty-three years, at about 5 p.m. on July 5th, 1900. She was said to have swallowed a large packet of sulphate of zinc at some time between 1 and 3 p.m. She was suffering from severe pain in the stomach and bowels, paleness of the countenance, coldness of the limbs, irregular pulse, and cold sweats, with purging, but had only vomited about a teaspoonful. I administered a large amount of carbonate of soda diluted freely with tepid water. Copious vomiting of a white fluid ensued (showing presence of carbonate of zinc), after which I directed that she should be frequently given white of egg and milk, and later on, when signs of collapse appeared, whisky with the latter. I injected ½ grain of morphine for the pain, and afterwards gave 1 grain opium tablets at intervals. I saw her again at 7 and 9 p.m. At the latter time she seemed easier. When visited at 9.20 a.m. on July 6th she was almost pulseless and collapsed. Every effort was made to revive her, but she died in a state of collapse at about 10.30 a.m., about twenty hours after taking the poison. *Necropsy* made forty-eight hours after death.

Externally.—The body was well nourished: *post-mortem* lividity slight, decomposition rapid. *Condition of the Head and Viscera.*—The membranes of the brain were congested, and there was a large amount of blood in the sinuses. The arteries at the base were congested, and there was a considerable amount of serous fluid in the left ventricle, and a little in the right. *Condition of Thorax and Viscera.*—The pleura was normal and non-adherent. Both lungs were engorged with dark blood. Heart normal. *Condition of Abdomen and Viscera.*—The stomach contained partially digested egg and milk. The mucous membrane lining it showed patches of intense inflammation, but this was much more marked in the small intestines, which were inflamed throughout. The inflammation in the large intestine was, again, of a patchy character. So vivid was the congestion of the small intestines that it was plainly visible from the outside before they were slit up. Liver 51 ozs., anæmic but otherwise normal. Spleen, soft and congested. Left kidney, 4½ ozs., and fatty; right, 4 ozs., normal.

Chronic Poisoning by Zinc

Batchelor and others[2] made a detailed clinical and laboratory study of twenty-four workmen exposed to inhalation of zinc in various forms. They found that zinc workers may absorb and excrete zinc in amounts considerably over the normal for years without the production of any symptoms or evidence of chronic poisoning whatsoever. They consider that the symptoms of chronic zinc poisoning described in the literature on the subject are due to the presence of toxic impurities in the zinc, such as lead, arsenic, cadmium, antimony, etc. Zinc is found in the tissues and excretions of most people on an ordinary mixed diet.[3]

Magnesium Salts

Source and Method of Occurrence. The sulphate and the two carbonates are used in medicine as the commonest and safest of the saline aperients. Toxic symptoms rarely occur after oral administration, but vomiting occasionally occurs, with some pain in the stomach. Cyanosis, stupor, and tetanic spasms were observed in a case recorded by Neale[4] in a lad *æt.* 15, who had taken an ounce.

The specific ion action of magnesium is seen when the salt is injected subcutaneously. It produces general paralysis of the central nervous

[1] *B.M.J.*, 1900, 2, p. 1706.
[2] *Jour. Indust. Hygiene*, Baltimore, 9: 322 (August 1926).
[3] *Journ. Biol. Chem.*, 72: 375 (1927). *Journ. Ind. Hyg.*, 8: 177 (1926).
[4] *Pharm. Jour.*, September 12th, 1896, p. 235.

system. Injected into the theca of the spinal cord it causes spinal analgesia. This action is antagonised by the intravenous injection of calcium. It is not seen when the drug is given by the mouth, owing to the great difficulty of absorption. Certain of the recorded cases of poisoning were no doubt due to impurities, such as arsenic.

Post-mortem Appearances. There are no special appearances.

Analysis. After filtration of the vomit or stomach contents the ordinary tests for magnesium may be applied to the filtrate.

Several delicate tests have been described, depending upon the adsorption of dyes on magnesium hydroxide. The most specific of these appears to be Titan Yellow. A drop or two of a ·61 per cent. aqueous solution of the dye is added to a few ml. of the solution to be tested, and the mixture is made alkaline with sodium hydroxide. In the presence of magnesium a red precipitate is produced.

Cases are rare, but the *B.M.J.*, 1891, 2, pp. 490 and 574, records two fatal cases of poisoning by Epsom salts. In one of these cases one ounce only of the substance was taken—an ordinary large dose.

Chas. Fraser, M.D., in the *Lancet*, 1909, 1, p. 1174, records a non-fatal case in a boy three and a half years of age from a "heaped-up teaspoonful." In the same article he refers to the fact that from 1841 to 1896 he could only find records of six cases, which he then proceeds to describe.

Poisoning by Silver Salts

Source and Method of Occurrence. The nitrate is the only salt that has caused symptoms of poisoning, and that usually by accidental slipping when the throat is being painted with "silver stick." It is used as a pure preparation and also in a form known as "mitigated silver."

Toxicity and Fatal Dose. The toxicity is not very great, but the irritation caused may be very severe.

In 1861 a woman, *æt.* 51, died in three days from the effects of taking a six-ounce mixture containing fifty grains of nitrate of silver (lunar caustic) given in divided doses.

Duration. In the case below the patient died in six hours, which is the shortest on record.

Symptoms. When taken internally silver nitrate causes acute gastro-enteritis with pain in the stomach, vomiting and purging, and death from collapse.

Silver used to be given for epilepsy over long periods, and in many cases produced a peculiar discoloration of the skin (*argyria*), but such cases are now practically unknown.

Treatment. Common salt is the best antidote, forming as it does a chloride of silver which is insoluble in acids, but requires evacuation by emesis or purging.

Post-mortem Appearances. Only characteristic of an irritant, *i.e.*, inflammation of stomach and intestines.

Cases. A well-marked case of poisoning with this substance occurred to Scattergood. A portion of a stick of lunar caustic dropped down the

throat of a child aged fifteen months. In spite of treatment, the child died in six hours in violent convulsions.[1]

Analysis. Stomach contents, vomitus, etc., will contain the silver precipitated as chloride. It may be extracted by digestion with ammonia, and reprecipitated as chloride from the filtered extract. The precipitate, heated on charcoal, is reduced to metallic silver, which dissolves in nitric acid. The solution of silver nitrate so obtained is submitted to test.

Tests. 1. Hydrochloric acid gives a white curdy precipitate of silver chloride, insoluble in nitric acid, but soluble in solution of ammonia.

2. Potassium chromate gives in neutral solutions a red precipitate of silver chromate.

3. Lime-water produces a brown precipitate of silver oxide.

4. Sodium phosphate gives a yellow precipitate of silver phosphate, soluble in nitric acid.

Poisoning by Barium Salts

Source and Method of Occurrence. The salts of barium are commercial articles. Insoluble barium sulphate is commonly used in X-ray examination of the bowels. The chloride and carbonate are present in some rat poisons. The salts are used in sizing cotton.

The substitution by mistake of a soluble salt such as the chloride for the insoluble sulphates for use in test meals for radiological purposes has resulted in a number of deaths.[2] A few deaths from suicide by taking the carbonates in the form of rat poison have also been recorded.

Toxicity and Fatal Dose. The soluble salts of barium are powerful poisons; they have not only a local irritant action, but have a special affinity for all kinds of muscle, in which they cause tonic contractions. They act as cardiac poisons, the ventricles of the heart after death being found rigidly contracted. They also paralyse the central nervous system.

The smallest single dose of a soluble barium salt known to have produced death in an adult is one hundred grains; one teaspoonful of the powdered chloride has caused death. Fühner,[3] however, has reported a death caused by 4·5 grams (72 grains) of barium chloride taken in two equal doses at an interval of twenty-four hours. Death occurred within twenty-four hours of the second dose. The poison had been supplied by a pharmacist in mistake for chloral hydrate.

Duration. This varies from one hour to seven days.

Symptoms. They usually consist of great weakness, salivation, thirst, colicky pains, excessive vomiting and purging. Speaking becomes difficult early in the case owing to paralysis of the muscles and swallowing becomes difficult from the same reasons. Paralysis of the muscles of the extremities follow, the paralysis being preceded by tonic and clonic convulsions.

Treatment and Antidotes. An ounce of sulphate of soda or Glauber's salts dissolved in a tumbler of water should be administered, with the object of converting the soluble barium salt into the insoluble sulphate of barium. The stomach tube should be afterwards employed, and the stomach thoroughly washed out. The after-treatment should be directed

[1] *B.M.J.*, 1871, 1, p. 527, and *Amer. Jour. Med. Sci.*, July, 1871, p. 287.
[2] McNally, W. D., *J. A.M. A.*, 84 : 1805 (1925).
[3] *Sammlung von Vergiftungsfällen*, 1930, vol. I.

to overcoming the depressant and convulsant action of the poison. The patient should be wrapped in warm blankets, and hot-water bottles applied to the feet ; brandy should be given either by the mouth, rectum, or hypodermically, to counteract collapse. If much pain persists, hypodermic injections of morphine should be employed.

Post-mortem Appearances. In one case, at the *post-mortem* examination made fifty-six hours after death, the stomach was found much ecchymosed from the fundus to the lesser curvature. The ecchymoses were in large patches over the fundus, in smaller patches towards the middle, and in still smaller patches at the pyloric portion of the organ. The intervening membrane was covered with a little mucus. Particles of the poison were found embedded in the mucous membrane. The peritoneal aspect of the stomach was of a pale red tint. There were numerous ecchymosed patches in the duodenum. Two grains of carbonate of barium were extracted from the stomach. The quantity taken could not be ascertained. Traces of barium were also detected in the liver. Intense inflammation of the lower bowel (rectum) is constantly observed.

Analysis. Where barium sulphate is suspected—*e.g.*, in vomitus or stomach washings after administration of sodium sulphate, the material is incinerated at a moderate temperature, and the ash is fused with sodium carbonate. The residue is then thoroughly extracted with water, and the insoluble part (which contains, as carbonate, any barium originally present) is dissolved in hydrochloric acid.

Otherwise the material may be dissolved in hydrochloric acid, and organic matter removed by means of potassium chlorate.

Chloride of Barium crystallises in thin plates : it is soluble in water. 1. The solution yields an insoluble white precipitate with sulphuric acid or a soluble sulphate. This precipitate is insoluble in nitric acid. 2. The powdered salt, burnt on platinum wire in a smokeless flame, imparts to it a greenish-yellow colour. 3. Ammonium carbonate gives a white precipitate. Redissolve this precipitate in dilute Hcl, neutralise the solution and place a drop on filter paper. Add a drop of 1 per cent. solution of sodium rhodionate. A red-brown colour is produced by barium or strontium (but not by calcium). The colour produced by strontium disappears on addition of HCl ; that due to barium becomes bright red.

Carbonate of Barium is a white insoluble powder. It is entirely dissolved with effervescence (carbon dioxide) by dilute hydrochloric acid. This solution on evaporation yields crystalline plates of chloride of barium, which may be detected by the tests mentioned above.

Note on the Tests for Barium.—It must be borne in mind that lead solutions also give a white precipitate with sulphuric acid, and that copper compounds give a more intense green colour to flame. The presence of these two metals must therefore be excluded by showing that the suspected solution when slightly acidified with dilute HCl gives no black precipitate with H_2S.

Cases. A woman, *æt.* 33, took by mistake for Epsom salts less than a teaspoonful (100 grains) of barium chloride. In half an hour there was a feeling of deadly sickness, with sharp burning pains in the stomach and bowels. Vomiting and purging set in violently, the purging being attended with straining. An hour and a half after she had taken the poison the following symptoms were observed : the face pale and anxious, eyes deeply sunk, surface cold, heart's action feeble, pulse scarcely perceptible, tongue natural, loss of muscular power, sensation and intelligence not affected, pupils natural. Fluids taken were instantly rejected with a ropy

mucus. There was pain in the stomach, singing in the ears, twitching of the face, and twisting of the legs and arms. In eight hours and a half the symptoms had abated, but in about fourteen hours the purging had returned, and the symptoms were much worse. There was a loss of voluntary muscular power. The breathing was slow and laboured, and indicated effusion in the bronchial tubes, but the woman was sensible. An hour later she was convulsed, and these convulsions continued in paroxysms for two hours, when she died, seventeen hours after taking the poison. During the fits she had several watery evacuations, and consciousness was lost. There was no *post-mortem* examination.[1]

A case of poisoning by this salt is reported by Wildberg. The symptoms were those of irritation, combined with an affection of the brain and nervous system. Giddiness, convulsions, and paralysis were remarked among them. In the case referred to half an ounce proved fatal in two hours ; in another instance one ounce, taken by mistake for Glauber's salts, destroyed life in *an hour*. In small doses, even, the chloride has been observed to affect the system powerfully. Orfila found the chloride of barium was absorbed : he detected it in the liver, spleen, and kidneys of animals poisoned by it.[2] A fatal case of poisoning by *nitrate of barium*, taken in mistake for sulphur, is reported.[3] Another fatal case is recorded in the same journal for June 1872, p. 1021.

A man employed in sizing cotton swallowed a portion about the size of a bean, thinking he was taking Epsom salts. He died in about fourteen hours. The symptoms were at first those of irritant poisoning, but in the later stage paralysis took place. The medical witness stated that he had found twelve grains of this salt sufficient to kill a dog.

In March, 1892, a female servant was given 150 grains of chloride of barium in mistake for Carlsbad salts. In twenty minutes she vomited. After two hours she was collapsed, with convulsive twitchings of the mouth, a rapid and feeble pulse, pain in the region of the stomach, and profuse watery diarrhœa. The vomited matter contained much blood. Death occurred in about two and a half hours. No *post-mortem* examination was made.

Carbonate of Barium is said to have destroyed life in two cases, in each of which only one drachm was taken ; but the following case shows that this compound is not so poisonous as the chloride. A young woman swallowed half a teacupful of the powdered carbonate, mixed with water, at a time when she had been fasting twenty-four hours. There was no particular taste. In two hours she experienced dimness of sight, double vision, singing in the ears, pain in the head, and throbbing in the temples, with a sensation of distension and weight at the pit of the stomach. There was also palpitation of the heart. After a time she complained of pain in the legs and knees, and cramp in the calves. She vomited twice a fluid like chalk and water. The skin was hot and dry, the pulse frequent, full and hard. These symptoms gradually abated, and she recovered, although the pain in the head and stomach continued for a long time.[4]

Two deaths from barium poisoning following X-ray examination were recorded in 1919—both due to the use of soluble barium salts in mistake for the sulphate.[5]

In one, a healthy woman, aged twenty-seven years, had gastro-intestinal symptoms. Examination with the X-rays after a meal containing sulphate of barium showed gastroptosis. Later, for a further examination, she was given the following prescription : Pure sulphate of barium 120 grammes : divide into three powders, one to be taken in a glass of water or milk at intervals of four hours. She took one powder in water and soon cried out, " It burns me ; I am poisoned." Vomiting of greyish fluid at once began and she went to stool and complained of severe abdominal pain. She died ten minutes after taking the powder. The remaining powders were greenish yellow and had a sulphurous odour. They were found to consist of sulphide of barium. In the second case an emaciated man, aged thirty-seven years, was given for radiological examination a pap containing 80 to 100 grammes of opaque salt. Having no carbonate of bismuth the pharmacist supplied carbonate of barium. The stomach appeared distended and slightly prolapsed. The gastric contractions were feeble and the meal did not pass into

[1] *Lancet*, 1859, 1, p. 211.
[2] " Ann. d'Hyg.," 1842, 2, 217.
[3] *Pharm. Jour.*, 1869, p. 181.
[4] *Med. Gaz.*, vol. 14, p. 448.
[5] *Lancet*, May 31st, 1919, p. 943.

the duodenum. On leaving the radiologist the patient suffered from malaise and felt that his abdomen was distended. An hour later he had violent pains with nausea. After another hour he vomited the greater part of the barium meal. Barium poisoning being suspected, he was given 25 to 30 grammes of sulphate of sodium and sulphate of magnesium. Barium was found in the urine four hours after ingestion. There was further vomiting and then a remission of symptoms for six hours, so that it was thought that the danger of poisoning had passed. But his general condition became worse, and the vomiting, at first bilious then with streaks of blood, recurred. He died thirty-six hours after taking the barium.

A female, *æt.* 28, finding herself pregnant, took an unknown quantity of carbonate of barium with suicidal intent. About 6 p.m. she vomited, and had severe pain in the stomach. She slept well, however, without further vomiting. Next morning after breakfast, at 7 a.m., vomiting was repeated ; nevertheless she walked to her situation about three miles distant, where she arrived about 9 a.m. She then looked pale and anxious, and complained of severe abdominal pain. She went to the privy repeatedly, either on account of diarrhœa or tenesmus. Nevertheless she attended to her duties as a domestic servant, but the vomiting and desire to go to stool persisted. At 2 p.m. she went to bed. At 4 p.m. she was found cold, pale, restless, and weak. There was no vomiting. At 8 p.m. she had alternate flushings and rigors. Speech was a mere indistinct whisper. Swallowing was difficult, and respiration laboured. At 3 a.m. she was distressed, the breathing was short ; she was perfectly conscious. She was found dead at 4 a.m., thirty-four hours after the commencement of symptoms. The exact time at which the poison was taken was not ascertained.

Poisoning by Mercury and its Salts

Source and Methods of Occurrence. Mercury is a metal used very largely in the arts, in commerce, and in medicine. Its production from mines is interesting, as the workmen engaged in obtaining it for commercial purposes suffer from chronic affections due to the metal.[1] It is peculiar amongst metals in that it seems to gain access to the body very easily, through the unbroken skin or through a sore, either by rubbing in of ointments or by exposing the body to an atmosphere in which mercury exists as a vapour (mercury can easily be boiled by an ordinary spirit lamp and vaporises appreciably even at ordinary room temperatures). Thus it happens that, however administered and in whatever combination, the specific effect of mercury on the body may easily be obtained.

At the Winchester Lent Assizes, 1859,[2] a quack was convicted of manslaughter by applying corrosive sublimate in powder to a cancerous tumour on the face. The man suffered from the usual symptoms. After death the bowels were found extensively inflamed and ulcerated. Corrosive sublimate was detected in the diseased part.

A girl, *æt.* 9, died from the effects of this poison, locally applied to the scalp for the treatment of ringworm. The liquid applied was alcohol containing eighty grains of corrosive sublimate to the ounce. She suffered from mercurial poisoning in a severe form, and died on the fifth day after the application.[3]

Acid nitrate of mercury has often been employed by accoucheurs as a local application in diseases of the neck of the uterus. In one instance in which it was thus used, the ordinary symptoms of mercurial poisoning showed themselves, and the patient appears to have suffered severely.[4] In another case the application

[1] *Vide* Oliver's "Dangerous Trades" ; Brouardel, "L'intox. mercurielle et en partic. l'intoxication professionnelle," *J. de méd. int. Paris*, 1903, 7, 194 ; Letulle, "Hydrargyrisme professionnel," *Bull. Soc. clin. de Paris*, 1888, 130 ; Koelsch, "Berufskrankheiten," Munich, 1926 ; Hamilton, "Industrial Poisoning in America" ; D. Hunter, "Industrial Toxicology," Oxford, 1944.

[2] *R.* v. *Crook.*

[3] See *Pharm. Jour.*, September 9th, 1871, p. 216 ; *Lancet*, 1871, 2, 413 ; and *Med. Times and Gaz.*, 1871, 2, 353.

[4] *Med. Gaz.*, vol. 45, p. 1025.

of the acid nitrate to the skin produced an eschar, and under the symptoms of mercurial poisoning caused the death of the patient on the ninth day. The mucous membrane of the stomach and intestines presented an arborescent redness, with patches of ecchymosis. Mercury was found in the liver.[1] In one case death took place with the usual symptoms from the external application of the nitrate in a liniment.[2]

The following forms of mercury and its compounds have been known to cause symptoms of poisoning :—

1. Mercury in metallic form ; simple medicinal forms.
2. Calomel.
3. Mercuric chloride ; corrosive sublimate.
4. White precipitate.
5. Red precipitate.
6. Cinnabar or vermilion.
7. Cyanide of mercury.
8. Mercuric sulphate.
9. Turpeth mineral.
10. Nitrates of mercury.
11. Sulphocyanide of mercury.
12. Organic compounds of mercury.

1. **Metallic Mercury** is not usually regarded as a poison, but may be a noxious thing. A large quantity of it in the fluid state may often be swallowed without affecting health, or without causing more uneasiness than that which may arise from its great weight. It may, however, when swallowed, produce salivation and the other constitutional effects of mercury. It rapidly passes through the bowels, and has in former days been used as a purgative in doses of an ounce or more. If the mercury is breathed or swallowed in a state of vapour, or if applied to the skin or mucous membranes in a state of minute mechanical division, in which state it appears to be easily susceptible of oxidation, it is liable to be absorbed, and to produce a poisonous action on the body. Symptoms of poisoning by the metal are seen in workmen engaged in trades in which they are exposed to the inhalation of mercurial vapours, for it must be remembered that mercury is appreciably volatile even at ordinary temperatures.[3]

Amalgams. Alloys of mercury with other metals are, of course, poisonous owing to their mercury content. The other metals present may also be toxic. Stock[4] has called attention to the danger of copper amalgams in tooth-stopping.

Blue pill and *mercurial ointment* are preparations in which mercury is finely divided, and probably, as in the mixture of mercury and chalk, more or less oxidised. Blue or mercurial ointment, which contains nearly half its weight of mercury, has attracted attention by reason of its poisonous effects on cattle. It is employed for the purpose of dressing sheep, in place of arsenic, and twenty-five tons of this ointment were sold in one year by a druggist in Boston, chiefly to farmers. Sheep poisoned

[1] *Edin. Month. Jour.*, 1864, p. 168.
[2] *Ibid.*, August 1864, p. 167.
[3] Giese, *Science*, 1940, 91, 476.
[4] *Med. Klin.*, 1928, Nos. 29 and 30.

with mercurial ointment have been sent for sale to the dead-meat markets in London. This is an (usually) unsuspected source of a noxious element in food for human beings.

Mercury with chalk is commonly regarded as an innocent medicinal compound of the metal, but if long kept and exposed a portion of the mercury passes to the highest state of oxidation, and thus produces a poisonous effect upon the system. This may account for the severe symptoms which have sometimes resulted from this preparation in medicinal doses. Duncan and Seely state that in one specimen which should have contained 37·5 parts of metallic mercury 4·05 parts had become converted into black mercurous oxide, and 22·25 parts into red mercuric oxide.

2. **Calomel** (*Mercurous Chloride—Subchloride of Mercury*). This substance, although commonly regarded as a safe mercurial compound, is capable of destroying life in small doses by causing excessive salivation with ulceration and gangrene, and in large doses by acting as an irritant poison.

3. **Corrosive Sublimate.** This substance has received a variety of chemical names. It has been at various times called *Oxymuriate, Chloride*, or *Bichloride of Mercury, Mercuric Chloride*, and *Perchloride of Mercury*. The modern chemical name is mercuric chloride, but because of its common use in medicine the old and popular name of *Corrosive Sublimate*, expressing the principal properties of the substance, is here retained. It is commonly seen in the form of heavy crystalline masses, or of white crystalline powder. Its *taste* is powerfully astringent and metallic, so that no poisonous quantity of it could be easily swallowed without a person becoming immediately aware of it. It is *soluble* in water, hot or cold, and speedily sinks in it, in which properties it differs strikingly from arsenic. One hundred grains of a cold saturated solution hold dissolved about seven grains of corrosive sublimate, and one hundred parts of boiling water (212° F.) will dissolve sixty parts of the poison. It is also readily dissolved by alcohol and ether.

4. **White Precipitate** (*Ammonio-Mercuric Chloride—Ammoniated Mercury*). In 1850, a woman who was indicted for administering this substance to her husband, owed her acquittal to the lenient assumption in her favour that it was *not* a poison. Out of fourteen collected cases in which white precipitate was taken in doses varying from a few grains to forty, two only proved fatal; and one of these was the subject of a trial for murder.[1]

5. **Red Precipitate** (*Mercuric Oxide—Red Oxide of Mercury*). This substance is poisonous, but instances of poisoning by it are rare.

The following case occurred at Guy's Hospital in 1833. A woman, *æt.* 22, who had swallowed a quantity of red precipitate, was brought in labouring under the following symptoms:—The surface was cold and clammy, there was stupor approaching to narcotism, frothy discharge from the mouth, and occasional vomiting: the vomited matters contained some red powder which was proved to be red precipitate. There was considerable pain in the abdomen, increased by pressure; and there were cramps in the lower extremities. On the following day the throat and mouth became painful, and she complained of a coppery taste. The treatment consisted in the use of the stomach-pump, and the free administration of albumen

[1] *R.* v. *Moore*, Lewes Lent Ass., 1860.

and gluten. She left the hospital four days afterwards, still under the influence of mercury. The quantity of oxide taken was not ascertained.

In 1797, a woman was treated in Guy's Hospital, who had taken red precipitate and laudanum. Severe abdominal pain, vomiting and bloody purging, were the most prominent symptoms. The woman recovered without salivation.

Vide also Mitchell, "A Fatal Case of Poisoning by Red Oxide of Mercury: Necropsy."[1]

6. **Cinnabar—Vermilion** (*Mercuric Sulphide*). The term *Cinnabar* is applied to a dark and heavy compound of sulphur and mercury, while *Vermilion* is the same substance reduced to a fine powder. It is well known as a red pigment, and is sometimes employed in colouring confectionery and wafers. It is stated to have proved fatal to animals in quantities of from thirty to seventy grains when applied externally to a wound. Cinnabar is sometimes used for giving a red colour to ointments, *e.g.*, sulphur ointment, and it is also employed as a colouring matter in vulcanised rubber for mounting artificial teeth. Although this insoluble compound of mercury cannot be regarded as an active irritant poison in the stomach, the placing of it in such a situation that it should be always in contact with the mucous fluids of the mouth is liable to lead to the usual consequences of chronic poisoning by mercury.

In 1864 a medical man reported the following circumstances. Upon the recommendation of a dentist, he had worn this red composition as a frame for false teeth, in place of gold. After some time he perceived a metallic taste in his mouth, the gums became inflamed and ulcerated, there was great weakness and want of nervous power, with pains in the loins and an eruption on the legs. When the composition was removed these symptoms abated. The substance contained a large quantity of vermilion; it had been mixed with the sulphur and rubber to give the appearance of the red colour of the gums. Wells had directed the attention of professional men to accidents of this nature. A patient of his, who had been provided with a frame of this description for the upper and lower jaws, perceived, soon after wearing it, a metallic taste in his mouth. His health failed, he lost his appetite and became emaciated: he suffered from flatulency, fœtid breath, and looseness of the bowels: his pulse was 100 and weak, and his tongue coated with a white film. This man was peculiarly sensitive to the action of mercury. He left off wearing the teeth, and became gradually better and stronger.[2]

7. **Cyanide of Mercury** (*Mercury Cyanide*). This substance is an active poison, and is not much inferior in activity to corrosive sublimate, but it has no corrosive properties. The toxic effects of mercury may be anticipated by a rapid and even fatal onset of cyanide poisoning.

8. **Mercuric Sulphate.** A case of suicide from this corrosive poison occurred in November 1891.

9. **Turpeth Mineral** (*Basic Mercuric Sulphate—Subsulphate of Mercury*). Fatal cases of poisoning by this compound are by no means common. Although insoluble in water, it is undoubtedly an irritant poison, and is capable of causing death in a comparatively small dose.

10. **Nitrates of Mercury.** These are corrosive poisons which are used for various purposes in the arts. They are solid white salts, easily dissolved by cold water when there is a little excess of acid present. Mercury poisoning in the felt hat industry is probably due to the presence in the air of fine fur which has been treated with mercury nitrate in the process of felting.

[1] *Boston M. and S. Jour.*, 1897, 136, 257.
[2] *B.M.J.*, 1863, 2, p. 266.

11. **Sulphocyanide of Mercury** is the chief constituent of the articles known as Pharaoh's serpents ; it is stated not to be very poisonous, yet in a case in which these gave rise to an action,[1] a lady ate a " serpent " by mistake and was very ill.

12. **Organic Compounds of Mercury** are formed as intermediates in various industrial processes which employ oxides of mercury as catalysts. Many of them are volatile toxic substances, and may cause chronic mercurialism among workmen who inhale their vapour.

Mercury fulminate is used for the manufacture of detonators and percussion caps, and apart from skin lesions of a characteristic kind, absorption from skin abrasions with symptoms of general mercury poisoning may be produced by exposure to the fine powder.

Methyl-, phenyl-, and tolyl-mercury compounds are largely used as fungicides, with special precautions in the manufacture and handling to prevent the occurrence of skin lesions (" mercury burns ") in the operatives. Long continued exposure to the fumes of diethyl mercury has been reported as causing death (*Canad. Pub. Health J.*, 1943, *34*, 158). Hunter et. al. recorded four non-fatal cases of poisoning from the inhalation of the vapour of methyl mercury compounds (substances not usually manufactured). The outstanding clinical findings were severe generalised ataxia, dysarthria and constriction of the visual fields without mental impairment (*Quart. J. Med.*, 1940, *9*, 193).

There are a great number of organic mercurial preparations on the market which are used principally as diuretics, usually by the intravenous route, and to a lesser extent intramuscularly. A number of deaths have been reported, especially with intravenous medication, death usually occurring suddenly.[2, 3, 4, 5, 6]

Toxicity and Fatal Dose. Mercury offers a remarkable illustration of the difference produced by chemical combination so far as toxicity is concerned. For instance, the difference between the amount of calomel and of corrosive sublimate that can be given without a likelihood of any unpleasant symptoms is very extraordinary ; three grains of the latter would almost inevitably entail death if not vomited, whereas three grains of the former is but a very ordinary medicinal dose. The soluble salts of mercury are naturally more active than the insoluble compounds, and are more corrosive than the salts of the other metals. This is due to the fact that the albuminate of mercury is readily soluble in excess of albumen or in the presence of sodium chloride. The fact that the mercuric salts are many times more active than the mercurous is due to their greater solubility.

Mercury has also the further peculiarity that it is easily volatilised and can in this state enter the body either by skin or lungs and produce deleterious effects.

Of the various preparations and salts of mercury the fatal doses of which have been recorded, the following may be cited :—

Of **metallic mercury**, 0·06 gram (1 grain) is said to have proved fatal when injected in the form of an emulsion ; on the other hand 27·2 grams

[1] *Vide Lancet*, 1, 1897, p. 605.
[2] Barker, M. H., and others, *Jour. Amer. Med. Assoc.*, 119 : 1001 (1942).
[3] De Grapy and others, *J. A.M. A.*, 119 : 998 (1942).
[4] Brown, G., and others, *J. A.M. A.*, 119 : 1004 (1942).
[5] Andrews, C. T., *B.M.J.*, 1 : 24 (1942).
[6] Tyson, Mary, *J. A.M. A.*, 117 : 998 (1941).

(over 400 grains) have been injected intravenously and the patient survived.[1]

The official dose of the metal is a half to three grains by the mouth, and a half to one grain by intramuscular injections.

The *smallest* doses of **corrosive sublimate** which have destroyed life when taken by mouth are *two* and *three* grains respectively. These were the cases of children; the quantities were accurately determined from the fact of its having been made up by mistake for calomel, which it was intended to prescribe.[2] It is probable that, in favourable circumstances, from three to five grains, or even less, would destroy an adult. Persons who had taken large doses have been known to recover when remedies were timely administered, or early vomiting was produced.

Lambert and Patterson report a case in which the patient died after the injection of one and a half grains ($HgCl_2$) intravenously.[3]

Pavy's experiments on dogs and rabbits show that **white precipitate** is a more formidable poison than it was formerly supposed to be. The greater number of recoveries were probably owing to the substance being early ejected by vomiting. Rabbits, which do not vomit, were killed by a dose of four or five grains in a few hours. After death, mercury was found deposited in various organs, but more in the kidneys than in the other viscera.

Of **turpeth mineral**, forty and sixty grains respectively have proved fatal, as have two successive doses of three grains each.[4]

Of **nitrate** of mercury a drachm has proved fatal, probably more due to the nitric acid than to mercury.

Of the **cyanide** twenty grains have proved fatal.

Mercuric salicylate has proved fatal in a dose of only $\frac{1}{3}$ grain.

These doses are given for what they are worth, but owing to the vomiting which occurs in human cases they tell us nothing as to the real amount necessary to kill after absorption.

There can be no doubt, too, that mercury is a drug for or against which many people have an idiosyncrasy. Thus, in 1881, Sir Thomas Stevenson was consulted respecting a lady whose life was brought into jeopardy by the administration of one dose of 1–32nd of a grain of corrosive sublimate, which is half the maximum official dose for internal administration. There were the symptoms of severe irritant poisoning and collapse, followed by salivation. It was clearly demonstrated by two independent analyses that the single dose of medicine contained only the 1–32nd of a grain of the poison. This is a very exceptional case.

Calomel in small doses has been known frequently to cause alarming gastro-intestinal symptoms.

Duration. The symptoms in acute cases come on within a very few minutes. They are rarely, if ever, delayed more than ten minutes, thus contrasting rather markedly with arsenic.

In an acute case, a person commonly dies in from one to five days; but death may take place much sooner or later than this. In the shortest fatal case on record, the quantity of poison taken was not ascertained, but the man died in less than *half an hour*. In a case reported in the

[1] Leschke, "Clinical Toxicology," 1933, p. 33.
[2] *Lancet*, 1845, p. 297.
[3] *Arch. Int. Med.*, November 1915.
[4] "Medicus," *Med. and Surg. Rep.*, 1884, 1, 93.

Edin. Month. Jour., 1860, vol. 1, p. 958, an adult who took from sixty to eighty grains of corrosive sublimate, did not die until the *twelfth day*. On the first day there was no complaint of pain in the gullet or stomach; the throat was painful on the second day, and the mouth and gums were affected on the third day. On the eighth day the man had apparently recovered, but he gradually became weaker and died on the twelfth day. In one case,[1] a large dose of corrosive sublimate (112 grains) caused death in *three hours and a quarter*. In another, which occurred in 1861, about five grains of corrosive sublimate caused death in six days; in both cases the poison was taken in solution.

Symptoms. In extreme cases there is a marked distinction between the symptoms of acute and chronic mercurial poisoning, though in many cases the symptoms of both forms occur together; in fact, if an acute case survives for more than an hour, salivation and other symptoms of mercurial absorption always appear.

Decrease of the chlorine content of the blood, increase in blood urea, and diminution of the alkali reserve are constantly found.

In the acute cases the particular salt swallowed seems to have no special relation to the symptoms produced. In the first place there is perceived a strong metallic taste in the mouth, often described as a coppery taste; and there is during the act of swallowing a sense of constriction almost amounting to suffocation, with burning in the throat, extending downwards to the stomach. In a few minutes violent pain is felt in the abdomen, especially in the region of the stomach, and increased by pressure, but this is occasionally absent. There is nausea, with frequent vomiting of long stringy masses of white mucus, mixed with blood, followed by profuse purging and painful tenesmus. The countenance is sometimes swollen and flushed, in other cases it has been pale and anxious. The pulse is small, frequent and irregular, and is scarcely perceptible when the symptoms become aggravated. The tongue is white and shrivelled—the skin cold and clammy, the respiration difficult; and death is commonly preceded by fainting, convulsions, or general insensibility. The external parts of the mouth, when examined, are swollen, and sometimes present an appearance as if the cavity had been washed with a solution of nitrate of silver; the lips are often swollen. The gums become inflamed and a general stomatitis occurs and increases in severity. Salivation occurs, the watery discharge rapidly acquiring a foul odour. The urine may be increased for a short period due to the diuretic action of mercury salts, but sooner or later suppression occurs from the extrusive damage to the kidney tubules. In many cases it may be one of the early symptoms. It occurred in a well-marked case in which the patient lived four days, but did not pass any urine during the whole of this time.[2] This symptom was observed in a youth, *æt.* 17, who swallowed three drachms of this poison, and died on the sixth day. During the last three days, no urine was secreted. The case was otherwise remarkable from the fact that no pain was experienced on pressure of the abdomen, and that the pulse underwent no change until shortly before death. In another case, in which twenty grains of corrosive sublimate in solution were swallowed, suppression of urine and salivation

[1] *Lancet*, 1862, 1, p. 119.
[2] Guy's Hosp. Rep., 1844, p. 24.

came on on the third day, and the patient died on the ninth day.[1] The rapidly toxic effect of mercury on the cells of the uriniferous tubules is one of its striking characters and constitutes a special danger in mercurial poisoning. As a general rule the earlier that anuria commences the more dangerous is the case.

The symptoms produced by **corrosive sublimate,** in the first instance, resemble those of cholera ; if the individual should survive several days, they are more like those of dysentery—violent straining, and mucous discharges mixed with blood, being very frequently observed. Irritation of the kidney, with all the clinical and chemical signs of acute nephritis and suppression of the urine even to complete anuria, is always to be expected, and is probably the usual cause of death.

A man, *æt.* 54, swallowed two-pennyworth of the poison (a quarter of an ounce) at 11 a.m. When seen, soon afterwards, he was on the bed in a state of great prostration ; his skin was blanched, and covered with a cold, clammy perspiration ; he vomited a thick, stringy, glairy substance. There was intense pain over the abdomen, and great purging with discharge of blood ; the pulse was scarcely perceptible, the tongue and the interior of the mouth were perfectly white from the local action of the poison. White of egg was given freely, and a mustard poultice applied to the abdomen. At one o'clock he was more depressed. At a quarter past two he expired. An inspection was made twenty-four hours after death. The external coat of the stomach was of a deep red colour. The mucous membrane internally had the appearance of a piece of dark crimson velvet. The intestines here and there were reddened. The large omentum for about an inch from the stomach was of a deep crimson hue. The other organs were healthy.

In another case in which five grains of the poison, dissolved in vinegar, were swallowed by a man, *æt* 25, the following symptoms were observed : Immediately after swallowing it, he felt a burning heat in his throat, and vomited freely. In two hours, there was great pain in the abdomen, he passed blood in his evacuations, and brought up a thick yellow frothy matter, tinged with blood. There was suppression of urine. He died on the sixth day. On inspection, the gullet presented marks of the local action of the poison. The mucous membrane of the stomach was reddened, and minutely injected throughout. There was no appearance of corrosion. The small intestines at their lower parts, as well as the large intestines, were deeply injected. The cæcum was but slightly affected. Seven ounces of the liver, and one-half of the stomach, yielded only minute traces of mercury. The greater part of the poison had been discharged by vomiting or by elimination during the six days which the patient survived. Thus, in spite of the removal of the poison from the stomach, the case may prove fatal. Eade reported a case in which a man swallowed a lump of corrosive sublimate : it was ejected from his stomach in about an hour. It was then smooth on the surface, and weighed about one drachm. The usual symptoms of mercurial poisoning followed, with suppression of urine. There was slight salivation on the fifth day, and the man died on the eighth day. Mercury was found in the liver.[2]

In the following case, reported in the *Med. Gaz.*, vol. 6, p. 315, it is probable that free nitric acid shared in the event. A boy dissolved some mercury in strong nitric acid, and swallowed about a teaspoonful of the solution. Soon afterwards he suffered excruciating pain in the throat, gullet, and stomach :—there was great anxiety, with cold skin, small pulse, colic and purging. He became gradually weaker, and died in about two hours and a half. On inspection, the throat, gullet, and stomach were found corroded and inflamed. Although he survived so short a time, the mucous membrane of the stomach was of a deep red

[1] *Lancet*, December 13th and 27th, 1845, pp. 650, 698.
[2] *Lancet*, 1870, 1, p. 303 (Hoffmann, Atlas, plates 41-44).

colour. In another case the application of mercuric nitrate to the throat as an escharotic, caused immediate death by asphyxia.[1]

At the Leicester Summer Assizes, 1857, a girl was charged with administering **nitrate of mercury** to her mistress.[2] The evidence showed that the accused had put the poison into some camomile tea prescribed for the prosecutrix. Only a small quantity was taken, as the tea had a nauseous taste. The symptoms were, a burning sensation in the throat and stomach, violent vomiting, and severe pain in the abdomen. The woman recovered.

With regard to **white precipitate**, the symptoms which it produces are violent vomiting, cramps, purging, and pain in the stomach, with convulsions. After death there is more or less inflammation of the stomach and bowels.

In 1877, a woman, *æt.* 30, was admitted into Guy's Hospital three hours and a half after having taken two-pennyworth, or "a good teaspoonful," of white precipitate. Within a few minutes of swallowing the poison vomiting and severe burning pain in the stomach came on. Twenty minutes after administration an emetic of ipecacuanha was given. On admission an emetic of sulphate of zinc was administered. The expression was anxious, the pulse and respiration were quickened: there was great thirst, severe vomiting, and great pain in the abdomen. The bowels acted frequently. She made a good recovery, and was discharged in nine days, no salivation having appeared.

In 1878, a man, *æt.* 66, took thirty grains of white precipitate. He vomited almost immediately. With the exception of a feeling of soreness in the throat his symptoms were not severe. In 1879, a third case, that of a woman, *æt.* 26, was admitted into Guy's Hospital. The patient had taken three-pennyworth of white precipitate in milk. There was severe burning pain in the region of the stomach, and a metallic taste in the mouth. She vomited on admission, and the vomit was found by Sir Thomas Stevenson to contain white precipitate. An emetic of sulphate of zinc and ipecacuanha was speedily followed by renewed vomiting. Purging set in next day. The woman recovered. Four days after the administration of the poisoning salivation appeared and continued for a few days.

Corrosive Sublimate Poisoning by Absorption from the Vagina

Holtermann[3] has recorded ten cases of poisoning from the effect of corrosive sublimate introduced in tablet form into the vagina. Amounts varying from 0·25 to 3 grams, inserted as anti-conceptional, anti-syphilitic, or abortifacient measures, or in one case accidentally, proved lethal (in from one to three weeks) in seven of these cases. Local necroses and ulcers were invariably present, especially in the posterior vaginal wall; in two cases they were so extensive as to lead respectively to vagino-colic fistula and to sloughing of the uterus. The symptoms of mercurial poisoning by absorption from the vagina do not differ from those of poisoning from the stomach or uterus or from intravenous injection; they consist chiefly of stomatitis, hæmorrhagic colitis, and nephritis, with terminal uræmia. Since the poisoning is due to absorption of mercury albuminate from the necrotic patches, immediate removal from the vagina of any remains of the tablets is of great importance. In a case described by Holtermann half of a one-gram tablet was inserted in the morning, and tenesmus, diarrhœa, and melæna occurred the same night. Albumen was found in the urine on the second day, stomatitis and

[1] Guy's Hosp. Rep., 1850, p. 206.
[2] *R.* v. *E. Smith.*
[3] *Zentralbl. f. Gynäkol.*, September 19th, 1925, p. 2133.

gingivitis were noted on the sixth day, and the patient, who had herself douched the vagina with salt solution on the appearance of the first symptoms, had completely recovered in three weeks.[1]

Chronic Poisoning

If mercurial salts are used with "devilish cunning" for homicidal purposes we may get a succession of mild attacks of the acute symptoms with remissions; such cases are, however, rare, and chronic cases more commonly arise from unintentional medicinal overdoses or from working amongst the vapours of mercury or the dust of its salts. There are colicky pains, with nausea, vomiting, general uneasiness, and depression. The salivary glands become inflamed and painful; the tongue and gums are red, swollen, and sometimes ulcerated, and there is fœtor of the breath. A deep blue line, somewhat like that observed in poisoning by lead, is sometimes found around the edges of the gums. The patient experiences difficulty of swallowing and breathing. The constitutional effects are indicated by looseness of the bowels, difficulty of breathing, spitting of blood, cough, general trembling or convulsive movements of the limbs and palsy, with slow fever and emaciation, under which the patient sinks. One of the most marked effects of slow or chronic poisoning by mercurial preparations is *salivation*, or ptyalism, indicated by an increased flow of saliva. This is by no means a necessary symptom in cases of acute poisoning by corrosive sublimate, but it not infrequently shows itself about the second or third day. In some instances the patient dies too rapidly for this effect to follow; but even when he survives some days, salivation is not always observed. Another result is the peculiar mercurial erethism, as it is termed, or affection of the nervous system. There is great mental excitability of the patient to external impressions. An unusual word disturbs him; and the question of the physician may cause him to grow pale, stammer, and become profoundly agitated. He is anxious without reason, sleepless, and disturbed by dreams. This condition after a variable period passes into the stage of mercurial tremor, or "trembles," as it is vulgarly termed. The tremor begins in the tongue, face, and arms, as a slight quivering, especially noticeable when the patient speaks, and it increases till convulsive twitchings are produced, extending over the whole body, and sometimes rendering locomotion impossible. The tremor disappears during sleep, and is increased by the slightest attempt at voluntary effort, or by mental emotion of any kind. If the patient is requested to hold out the hand or to protrude the tongue, he does so in a jerky manner; and attempts to walk result in the most grotesque contortions. As mercury may be easily detected in the saliva by a process to be described, the discovery of the metal in this fluid will show the real cause of the salivation.

A man, *æt.* 32, suffering from chronic poisoning by the nitrate of mercury, was admitted into Guy's Hospital in 1863. He had been for four years engaged in packing the fur of rabbits, rats, and other animals, the dried skins of which had been previously brushed over with a solution of nitrate of mercury. For the first three years he suffered only from a feeling of general weakness. For about a twelvemonth he could not hold his hand steadily enough to shave himself, and soon afterwards he completely lost control over the voluntary movements of his limbs. Three or four months before his admission he had had slight twitchings of his

[1] *B.M.J.*, Epitome, October 24th, 1925.

muscles when in bed. He was not at all emaciated. He said he had been salivating for about three months, soon after he began his occupation of packing furs ; but his gums were not tender, and he had no metallic taste in his mouth. A month before his admission he gave up his work. When he became a patient he could walk with assistance, but on standing or lying down he could not control his limbs, which trembled considerably. There were continued involuntary movements of his body and limbs, like those of chorea. He became much exhausted, owing to want of sleep, and perspired profusely. The urine was highly coloured, but otherwise natural. Twelve ounces of it did not yield any mercury. No treatment appeared to give him rest or relief. Chloroform arrested the spasmodic movements, but only while he was under its influence. In five days he passed his urine involuntarily. He was quieter and slept a little at night. He had difficulty in swallowing, became gradually weaker, and died, apparently from exhaustion, a fortnight after his admission. On inspection, the body was well nourished ; the muscles were firm and healthy. The brain and spinal cord were found to be quite healthy. The lungs, heart, liver, spleen, and kidneys were free from any morbid appearance, or any change to indicate a cause of death.

An analysis was made of the brain, liver, and kidney. Six ounces of each organ were dried, and one-half of the dried residue, treated with hydrochloric acid and water, as elsewhere described, gave, in forty-eight hours, on a small portion of copper-gauze, a greyish, white deposit, which yielded globules of metallic mercury when heated. The kidney yielded the largest sublimate ; but the quantity obtained from each organ was small, and might be described as mere traces. In the fur similar to that which the man had been engaged in packing a small quantity of a soluble salt of mercury was readily detected. The case, which at first presented some difficulty in accounting for death, thus resolved itself into one of exhaustion as a result of chronic poisoning by mercury in somewhat unusual circumstances. It is probable that the man received the dust of the dried nitrate through the air which he breathed, as well as by contact with his mouth, nostrils, and skin. As other workpeople similarly engaged were not found to have suffered, this may have been a case of mercurial poisoning due to idiosyncrasy.

Similar but milder symptoms are frequently seen when mercury is being administered by inunction ; they speedily subside on cessation of the treatment, but the teeth may be lost if inunctions are persisted with.

Treatment of Acute Poisoning

The prognosis is unfavourable, especially if retention of urine occurs.

The stomach should be washed out immediately by the stomach tube with warm water, and albumen in the form of white of egg or milk administered. Skimmed milk is better than whole milk for the purpose.

If the administration of the albumen is not at once followed by emesis, the stomach tube must again be used, for the albuminate formed is not insoluble. This must be continued. Animal charcoal should be given with the object of absorbing the poison. Two tablespoonfuls of medicinal charcoal should be suspended in a pint of warm water and introduced into the stomach as soon as possible.

General treatment must be adopted to combat the shock and pain, and particular attention must be given to the kidneys.[1] Treatment of the acute nephritis, which is almost inevitable, is most essential. The excretion of the salt by the caecum and bowel and by the saliva must be

[1] Peters *et. al.*, *Amer. J. Med. Sci.*, 1933, *185*, 149.

kept in mind, and the bowel irrigated and the mouth kept clean. Intravenous injection of normal saline with sodium bicarbonate should be made to counteract the lowering of the alkali reserve of the blood. Intravenous injection of glucose (20 c.c. of a 20 per cent. solution) and a similar quantity of sodium chloride has been recommended. Intravenous injection of sterile solution of sodium thiosulphate has been recommended by many observers. This is given in doses of half a gram once a day. There seems to be a certain amount of evidence that this salt is of a certain value in arsenical dermatitis, and its value in mercurial poisoning has also been vouched for. Dennie and McBride,[1] Semon,[2] Kuhn and Reese[3] and many other writers claim that it has a definite value. We have not found that it has the specific effect claimed for it, and Haskell and others,[4] from their experimental observations, have come to the conclusion that it is of no value. However, it is devoid of bad effects, and may be given a trial (see "Arsenic Poisoning"). Sodium formaldehyde sulphoxylate has been recommended[5] and good results claimed,[6] though others have reported adversely.[7] It is stated to reduce mercuric compounds to the less toxic mercurous form.

The development of "B.A.L." (i.e. British Anti-Lewisite or 2 : 3 Dimercapto propanol) has introduced a new and potent agent into the treatment of poisoning by certain of the metals, including mercury. The mode of action of this compound is more fully considered in the section on Arsenic poisoning (see p. 417), but cases have already been reported of its successful use in mercury poisoning, including acute poisoning by mercuric chloride. For such acute cases, very large doses have been used, viz. 300 mgm. intramuscularly as an initial injection, followed within twelve hours by two or even three further injections of 150 mgm. each. These are very large doses indeed, liable of themselves to cause toxic symptoms, but they appear justified by the desperate condition of the victims of mercury poisoning, and by the successes recorded. The optimum doses will no doubt be more definitely established by further experience, but there appears no doubt that in "B.A.L." we have a new and valuable agent for combating, amongst other intoxications, the dire effects of mercury poisoning, provided it is employed early enough.

Post-mortem Appearances. These, as in the case of arsenic, are chiefly confined to the stomach and bowels. Corrosive sublimate, however, affects also both the mouth and throat ; the mucous membrane is softened, of a white or bluish-grey colour, and sometimes inflamed ; that which lines the gullet is similarly affected, partly corroded and softened. The mucous membrane of the stomach is more or less inflamed, sometimes in patches ; and there are masses of black extravasated blood found beneath it. Occasionally it has a slate-grey colour, and the mucous coat beneath may be found reddened. A case occurred in Guy's Hospital, in which the mucous membrane was simply inflamed : it much resembled the condition presented in cases of arsenical poisoning. The coats of the stomach

[1] *Arch. Derm. and Syph.*, January, 1923, and *Jour. Amer. Med. Assoc.*, December 27th, 1924.
[2] *B.M.J.*, April, 1924.
[3] *Jour. Amer. Med. Assoc.*, December 5th, 1925.
[4] *Ibid.*
[5] Rosenthal, *Pub. Health Rep.* 1933, *48*, 1543.
[6] Idem, *Jour. Amer. Med. Assoc.*, 1934, 102, 1273.
Monte & Hill, *Ibid*, 1940, *114*, 1433.

are sometimes corroded, and so much softened that the organ cannot be removed from the body without laceration. Similar appearances have been met with in the small and large intestines, and especially in the cæcum.

The changes in the large intestine are often very striking and are of a dysenteric character with necrosis, which spreads, and, in the event of the individual living some time, may involve practically the whole of the lower bowel. This "mercuric dysentery" is due to a secondary excretion of mercury into the bowel.

In a case in which a man died forty hours after having swallowed 120 grains of powdered corrosive sublimate, the mucous membrane of the stomach, duodenum, upper portion of the ileum, and parts of the large intestines, were found of a bright red colour. This appearance was most marked at the cæcum and sigmoid flexure of the colon. The local action of the poison on the mouth and throat was in this instance considerable.[1] Perforation of the stomach is rare as an effect of this poison; there is, however, a case on record. Appearances like those just described have been seen in the alimentary canal, not only where the case has terminated fatally in a few hours, but where it has been protracted for six, eight, and even eleven days. In the case of a man, *æt.* 42, who swallowed, by mistake, thirty grains of corrosive sublimate in solution, and who died on the twelfth day, the stomach was found empty and the mucous membrane was of a dull, dark-red colour, chiefly about the smaller curvature. This organ was softened, and near the intestinal end was grey, pulpy, and gangrenous. In the gullet, the lining membrane appeared to have been stripped off in shreds. The intestines were in a state of intense inflammation, passing into gangrene. The other viscera presented nothing abnormal. In this case the symptoms were manifested in a few minutes: there was a burning pain extending down the gullet to the stomach, described as if the parts were on fire; there was no mark of corrosion in the mouth; there was a sensation as if the throat were closed; and there was blood in the vomited matters as well as in the evacuation. There was no salivation at any period.

The kidney is often severely damaged. Since Salkowski first showed the presence of lime infarcts in the straight tubules of rabbits poisoned by sublimate, this condition has been extensively studied experimentally and in man. Cloudy swelling and necrosis of epithelium with consequent lime deposits may be very striking.

Unless appearances of acute irritation or changes in the kidney such as the above are present, it is impossible without analysis to ascertain that mercury has caused death, for it leaves no other visible trace in the tissues.

Chemical Analysis. A person may die from the effects of corrosive sublimate, **and no mercury may be found** in the tissues. A case of this kind occurred at Guy's Hospital; and another, in which deceased died in fifteen days from a large dose of corrosive sublimate in whisky, has been reported by Geoghegan. On this occasion, although the local effects of the poison on the throat, stomach, and bowels, were of an intense kind, the viscera, on careful analysis, yielded no trace of mercury; the metal had been entirely eliminated in fifteen days.[2] *Vide ante*, p. 246.

[1] *Edin. Month. Jour., December* 1851, p. 532.
[2] *Med. Gaz.*, vol. 46, p. 253.

In Organic Liquids. A sample of the liquid should first be examined for mercury by the **Reinsch test**, *vide* under "Arsenic." The liquid should then be filtered from any insoluble matter. The latter should be pressed, dried, and set aside for a separate analysis. Any heavy sediment may be obtained by decantation, dried, weighed, and separately examined. In the investigation of the solid matter, insoluble compounds of mercury are, of course, to be sought. The colour may be of service (*e.g.*, vermilion, red precipitate). Calomel (or other mercurous salts) will turn black on addition of ammonia. For the final confirmation or estimation of mercury, organic matter may be destroyed by the potassium chlorate method or by boiling with concentrated nitric acid.

For the detection of mercury in the clear filtrate a small electrolytic couple, made by twisting a layer of gold-foil round a layer of zinc-foil, may be introduced. The liquid should be slightly acidified with hydrochloric acid and warmed. The couple should be suspended in the liquid for some hours. If mercury is present, even in small quantity, the gold will sooner or later lose its colour and become "silvered," while the zinc will be wholly or in part dissolved. The slip of gold-foil may be washed in water, and afterwards in ether, and dried. Alternatively a small piece of fine copper gauze may be suspended in the solution for some hours (*e.g.*, overnight), removed, washed, and dried in the same way. The gold-foil or copper gauze should be divided into two equal portions. One should be submitted to heat in a tube, when globules of mercury will be obtained: the other in the case of gold, should be heated with a few drops of concentrated nitric acid, until the gold has reacquired its yellow colour.

Most of the absorbed mercury is excreted in about six days, but traces may be detected in the excreta for months.[1]

The tube in which the foil or gauze has been heated should be carefully examined under the microscope, using first the low power and later, if necessary, a high power. Attention should be paid particularly to scratches on the inside of the glass tube, as the mercury globules tend to be deposited along these in chains. The sublimate of mercury is quite unlike anything else; the perfect sphericity of the globules, their silvery whiteness by reflected light, their opacity by transmitted light, and the fact that reheating merely causes them to re-sublime without change, identifies it quite definitely. The sublimate is soluble in a mixture of nitric and hydrochloric acids or in hot concentrated nitric acid, the solutions, on evaporation, depositing crystals of mercuric chloride and nitrate respectively.

The nitric acid solution from the gold-foil similarly leaves a residue of mercuric nitrate on evaporation.

For the further confirmation of the presence of mercury, redissolve the solid mercuric chloride or nitrate in a little water and apply the following tests:—

(1) Add a drop of very dilute potassium iodide solution. Mercuric salts give a scarlet precipitate which dissolves in excess of potassium iodide. Addition of sodium hydroxide and a trace of an ammonium salt then gives a yellow colour or precipitate.

(2) Sodium hydroxide solution gives a yellow precipitate; ammonium hydroxide a white one.

[1] *Arch. Dermat. Syph.*, 1935, 32, 1.

(3) To a drop of the solution in a small test-tube or on a filter paper add a drop of fresh alcoholic solution of diphenylcarbazide; mercuric salts give a violet-blue precipitate.

Even though the gold-foil used for the separation of mercury does not become appreciably changed in colour, the further tests described above should always be carried out. They are sufficiently delicate to detect quantities of mercury which are incapable of concealing the yellow colour of the gold-foil.

For the investigation of mercury poisoning in a living person, saliva should be examined as well as, of course, urine and fæces. Although mercury is not a "normal" constituent of the human body, so that any found in the tissues or excreta must have come from external sources, the diagnosis of mercury *poisoning* must rest on more than chemical analysis. The history and symptoms of the case must be taken into account, for many medicinal preparations contain mercury compounds.

Young and Taylor[1] have described an electrolytic method for the determination of mercury in tissues and body fluids. They claim to estimate quantities of the order of 5–10 mg. with a maximum error of 5 per cent. The material for analysis is digested (*e.g.*, 250 c.c. urine, 10 grams tissue or fæces) under a reflux condenser with a mixture of sulphuric (2 c.c.) and nitric (25 c.c.) acids with addition of potassium permanganate (2 grams). From the resultant colourless fluid, the mercury (possibly with other metals—*e.g.*, manganese) is deposited electrolytically on a platinum cathode. With a current of 0·5 amps. at 6 volts, deposition is complete in about twelve hours. The electrode is then washed with distilled water and the deposit is dissolved in hot fuming nitric acid (5 c.c.). The extract is diluted to 100 c.c. with distilled water (all reagents from this stage onwards must be chloride free), a drop of potassium permanganate is added to oxidise any mercurous compounds, and the excess permanganate is destroyed by adding 3 per cent. hydrogen peroxide drop by drop till the colour disappears. Five cubic centimetres of 10 per cent. ferric ammonium alum solution are then added, and the solution is titrated with 0·05 or 0·01 N potassium thiocyanate solution until the first rose tint appears. A "blank" analysis must be made on each batch of reagents.

The identification of the actual compound of mercury will usually rest on examination of undissolved material or, the finding, in fluid stomach contents, of an acid radicle which, but for the accompanying mercury, would not be expected. Thus finding, in such contents, mercury and chloride would not be proof that corrosive sublimate had been administered, but finding mercury and nitrate would be good evidence of the ingestion of mercury nitrate.

Corrosive sublimate forms opaque silky prisms, soluble in water (easily so in hot water), alcohol, and ether. Besides the tests already given for mercury, it (as do other soluble mercuric salts in sufficient quantity) gives a grey precipitate of mercury with stannous chloride, and a black precipitate of mercuric sulphate with H_2S or ammonium sulphide. It gives the tests for chlorides.

The *nitrate*, when heated with dry carbonate of sodium in a tube, yields a sublimate of mercury. It also responds to the usual tests for nitrates.

[1] *J. Biol. Chem.*, 1929, 84, 376.

Calomel is distinguished from corrosive sublimate by its insolubility in water, alcohol, and ether; and from white precipitate by its being blackened by alkalies. A sublimate of metallic mercury may be obtained from it by heating it with carbonate of sodium.

Vermilion, or an organic mixture containing it, may be entirely decomposed by nitro-hydrochloric acid. The residue, evaporated to dryness, contains corrosive sublimate. This may be taken up by water and the usual tests applied. Ether will separate corrosive sublimate from the aqueous solution. The vermilion contained in vulcanite is thus readily detected. Red sulphide of mercury is not blackened, like red lead, by sulphide of ammonium, and is not dissolved by hydrochloric acid, like red oxide of mercury. It yields potassium sulphide and globules of metallic mercury when heated with cyanide of potassium.

White precipitate is a heavy, insoluble, chalky-looking substance, containing about 80 per cent. of mercury. In commerce it frequently contains corrosive sublimate to the amount of 1 or 2 per cent. It is not used internally, but it is employed in the treatment of skin diseases. It is soluble in acids, is not blackened by alkalies, and yields a mercury sublimate when heated with carbonate of sodium. It is not dissolved by water, but becomes yellow by long boiling. If boiled in a solution of potash, it evolves ammonia, and yellow mercuric oxide is precipitated. It may be detected in organic fluids and solids by boiling them in one part of hydrochloric acid and four parts of water. The mercury may then be separated by means of copper.

The colour of *red precipitate* and the fact that when heated in a close tube it is resolved into oxygen, and mercury which is deposited in globules, are sufficient to identify it.

A typical case of mercurial poisoning and its treatment has been reported by Hayden Ellis :—[1]

A man aged 20 years took three grams of corrosive sublimate dissolved in a glass of water. Half an hour later he had severe vomiting and diarrhœa with severe abdominal pain. Blood was present in both vomit and fæces. Three hours later blood appeared in the urine. He was given white of egg in milk and removed to hospital.

He continued to have bouts of colic followed by the passage of blood clots and mucus per rectum about every half-hour. Vomiting continued to be frequent, and the vomit consisted of bile and blood-clots. The mental condition was clear. Abdominal movement was restricted; there was diffuse tenderness, and gentle palpation was followed by violent bowel action and much pain. There was also some tenderness in the loins. His blood-pressure was 152/74.

On the second day pain, diarrhœa, and vomiting continued; stools were passed at the rate of one an hour and were darker in colour. During the day the patient passed 5 ml. of urine containing a small quantity of albumin. The centrifuged deposit showed occasional red blood-cells, white cells, and many hyaline casts. Kaolin poultices were placed over the loins. Water and sips of iced orangeade were taken. Restlessness and pain were treated with morphine, ¼ gr. four-hourly. Frequent vomiting continued, and much fluid was lost in the fæces. An intravenous drip was started in the left arm, and fluid was given at the rate of 4 pints (2·27 litres) in 24 hours—namely, alternating pints of dextrose-saline (4.8 per cent. and N/5) sodium sulphate (4·2 per cent.), and normal saline.

By the third morning the patient was very drowsy and there was twitching in the limbs. The tongue was covered with brown fur, and he complained of severe headache. Morphine, ¼ gr. four-hourly, and the intravenous drip were continued.

[1] Ellis, R. H., *B.M.J.*, 2 : 197 (1946).

He vomited 1 litre during the day, but passed no urine. There was little improvement over the next three days, though the abdominal pain was less and the stools not so frequent. No urine had been passed since the original 5 ml. after admission. The patient was now mentally alert and worried about himself. Blood urea was 236 mg. per 100 ml., and the patient was very ill. All veins except one had now been utilised, and the intravenous administration of fluid was discontinued. On the seventh day the abdominal tenderness had disappeared, the diarrhœa and vomiting had improved slightly, but the patient was hiccuping continuously. The plantar responses were extensor. Blood-pressure, 174/84; blood urea, 350 mg./100 ml. Morphine was changed to heroin, $\frac{1}{8}$ gr. (8 mg.) four-hourly, as this was thought less likely to aggravate vomiting.

On the morning of the eighth day the patient's condition was critical. No urine had been passed since the second day (5 ml.). Intravenous therapy had been stopped for 24 hours on account of technical difficulties, and was begun again—namely, dextrose-saline at the rate of 4 pints (2·27 litres) in 24 hours. During the day and following night 1·85 litres of urine was passed, and the patient's condition improved. The next day there was a pronounced improvement: 1.9 litres of urine, of specific gravity 1010, was passed; twitching and headache abated; and the patient was able to take rusks and 1·7 litres of fluid by mouth. Blood urea, 300 mg./100 ml.; hæmoglobin, 65 per cent.; W.B.C. 21,000.

Over the course of the next three days intravenous dextrose-saline was continued and the diuresis and general improvement were maintained. Vomiting was less, and the patient was able to take solid food in the form of eggs and custard, calves'-foot jelly, rusks, and pounded fish. At the end of this time the last vein had thrombosed, and the drip was discontinued. Blood urea, 380 mg./100 ml.; plasma chlorides, 419 mg. NaCl/100 ml.; alkali reserve, 42 vols. CO_2 per cent.; Blood-pressure, 150/60. The heroin had been discontinued and phenobarbitone, $\frac{1}{2}$ gr. (32 mg.) four-hourly, was given in its place. In spite of the high blood-urea figure the patient felt well and was cheerful.

On the 17th day after admission the blood-urea was 170 mg./100 ml.; plasma chlorides, 487 mg. NaCl/100 ml.; alkali reserve, 52 vols. CO_2 per cent. The centrifuged deposit of each specimen of urine passed showed successively diminishing numbers of red and white blood cells, and all showed persisting hyaline and granular casts. On the 24th day the patient was his normal self, eating well, with no vomiting, and passing one semi-solid darkish stool per day. The urinary output was in the region of 2 litres in 24 hours; the blood urea was down to 63 mg./100 ml.; plasma chlorides had been raised to the normal level of 576 mg. NaCl/100 ml., helped by the giving of 5 g. of NaCl daily in a litre of lime-juice; alkali reserve, 56·7 vols. CO_2 per cent.; blood-pressure, 148/63. The urine contained no albumin and the centrifuged deposit was normal.

The following case, arising from the *external application* of corrosive sublimate, is interesting.[1] It shows some unusual symptoms, probably not due to the mercury, but to shock and fear:—

A. B., a dispenser, aged twenty-nine, dissolved 60 grains (or more) of corrosive sublimate in hot water (5 v.) intending to make a bath for an eruption which he believed to be scabies. He sponged his arms, abdomen, thighs, and scrotum with the warm fluid, and then went to attend to people in the shop. A smart tingling came on at once, which, when he had attended to three customers, became severe pain, principally in the penis and scrotum.

When seen half an hour after the application, the penis and scrotum were red and much swollen, and there was a papular rash on the groin and slightly on the abdomen. He was suffering very severe pain, was twitching, and jerky in his speech. Morphine, $\frac{1}{4}$ grain, and cocaine, $\frac{1}{4}$ grain, were given hypodermically, the body sponged with flour and water and egg albumen, and the whites of two eggs given in tea. There was difficulty in swallowing the second cup (one hour and a half after the application). At the end of another hour he was worse in every way, could hardly speak, swallowed with the greatest difficulty; the left hand was clawlike and stiff, and there were severe tremors. He complained of burning of his lips, throat, and stomach. Four hours later he retched violently, but with

[1] *B.M.J.*, 1903, 2, p. 1212.

little result, and had a severe general clonic spasm lasting about twenty seconds. The spasma recurred about every ten minutes, and did not seem to be induced by outside stimulation. Twice marked opisthotonos occurred. During the attack there was absolute general rigidity, inability to breathe (lasting once for ninety seconds); the pulse, which between the spasms was about 100, became running in character. There was an expression of terrible fear. A general tremor preceded each attack, with an elevation of the shoulders. Rigidity was partial during the intervals, and the breathing was shallow and mostly abdominal. The symptoms were all at their height five hours after the application.

Two hypodermic injections of brandy, ♏ xx., and morphine, ¼ grain, were given. The patient was surrounded by bottles full of hot water wrapped up in bath towels wrung out of boiling water. Amyl nitrite and chloroform were administered with good result, though the shallowness of the breathing delayed it somewhat. The rigidity between the attacks diminished, but when after two hours' administration chloroform was stopped, another rather sharp, though not prolonged, spasm came on. Chloroform was then given for three more hours, and stopped nine hours after the application of the poison. The case then went on well. For two days there was a burning sensation, and one or two attacks of sickness, but there was no salivation, no suppression of urine, and no purgation. Large grey blisters covered the scrotum. There was marked weakness of the left hand, which improved every day—as did the inability to protrude the tongue, which had appeared early in the case.

The previous history probably accounts for the unusual symptoms, the patient having been wounded in the late war, and spending seven months in hospital with paralysis of the left side, from which he had completely recovered.

Poisoning by Copper and its Salts

Source and Methods of Occurrence. The two most commonly known salts of copper are the sulphate, or blue vitriol, and the subacetate, or verdigris. The former has been taken and administered in large doses, for the purpose of suicide and in attempts at murder.

In 1886 a man was convicted of attempting to murder his wife by administering to her sulphate of copper in spruce and peppermint water. He was sentenced to twenty years' penal servitude.[1] In 1884 a girl was convicted of poisoning her mistress by introducing sulphate of copper into a jug of beer. The taste of the substance was perceived; the only result was severe vomiting.[2]

Copper is not very adaptable to secret murder, owing to the colour and the strong metallic taste possessed by the salts. This would in general render it impossible that the poison should be taken unknowingly. It has, however, been given for the purpose of procuring abortion. In doses of half an ounce and upwards it acts as a powerful irritant on adults, and a much smaller quantity would suffice to destroy infants or children. There are two copper salts—the arsenite (Scheele's Green) and the aceto-arsenite (Brunswick Green)—which owe their poisonous properties to arsenic. These will be elsewhere considered. The chloride and carbonate are also irritant poisons.

With the exception of these salts, copper poisoning may occur from the contamination of food stored or cooked in copper containers which are not kept clean or from the ingestion of food to which copper has been added in order to keep the green colour of vegetables.

Metallic copper undergoes no change by contact with *water* unless oxygen is present, when a hydrocarbonate is formed. If the water contains an acid such as vinegar, or common salt, or if there is oily or fatty matter in contact with the metal, then the copper is more rapidly oxidised.

[1] *R.* v. *Reynolds*, C. C. C., September 1886.
[2] *R.* v. *Mary Baker*, C. C. C., October 1884.

and the liquor or fat acquires a green colour. If the **copper vessel** is kept perfectly clean, and the food prepared in it is allowed to cool in other vessels, there is not much risk of its acquiring a poisonous impregnation. Under the influence of heat and oxygen, a portion of copper becomes dissolved, and the oily or other liquid acquires a green colour. The preparation of fruits, such as preserves, in copper vessels is necessarily attended with some risk, for on cooling a green crust is apt to form on the copper, just above the line where the air and acid liquid meet. Some liquids, while boiling, appear to be little liable to this impregnation ; thus, coffee, beer, milk, and tea have been separately boiled for two hours, in a clean copper vessel, without any portion of the metal being taken up by any of the liquids. Accidents traceable to the kitchen are usually prevented by lining the copper vessel with tin, but in very large boilers this plan is not always adopted ; cleanliness alone is trusted to, and this, when properly observed, is a sufficient preventive. In reference to tin-lined culinary vessels the tin is often worn away, and the corroded copper is thus exposed to the action of any acids contained in the food.

In 1866 some rhubarb-stems were stewed in an imperfectly tinned and dirty copper vessel, and were supplied to a family for dinner. The children and their governess partook of it—the latter very freely. All were taken ill. The governess suffered most ; there was violent sickness, and other symptoms of irritation. She partially recovered under treatment, but had a relapse, and died from the effects of the poisoned food.

The oxalic and malic acids in the vegetables probably dissolved compounds produced from the copper by exposure to damp air.

The tin used for lining copper vessels is frequently alloyed with a large proportion of lead, and thus lead-poisoning may be substituted for poisoning with copper. It is now well recognised that some cases of alleged poisoning by copper are really due to the arsenic which is almost universally contained in commercial copper.

In the **making of preserved fruits and vegetables**, the salts of copper are used in some countries for the purpose of giving a rich green colour. Many of the green pickles sold in shops were formerly thus impregnated with the vegetable salts of this metal, to which they owed their bright grass-green colour. The quantity of the copper contained in such articles may not be sufficient to cause fatal effects ; but symptoms of gastric irritation are sometimes alleged to arise from the consumption of such vegetables and pickles ; no evidence of the truth of the allegation has been produced, and the use of copper for colouring vegetables is allowed in many countries.

According to Tschirch, the practice with some manufacturers is to treat the green vegetables with a dilute solution of sulphate of copper, and afterwards wash them thoroughly with water before boiling. The result is the formation in the plant tissue of phyllocyanate of copper, a compound containing 7·35 per cent. of copper, insoluble in water, dilute hydrochloric acid, or acetic acid, but soluble in alcohol. The tinctorial power of this compound is said to be very great, and the colour is unaltered by light.

Considering that copper itself is not an active poison, and that this compound contains it in so small a proportion and in so insoluble a form, Tschirch expressed the opinion that this treatment of preserved vegatables might be allowed. Kobert, on the other hand, stated it was true that a

large dose of a copper salt might be taken at one time without producing toxic effects, but it had not been proved that a continuous administration would not produce chronic poisoning. Moreover, although it might not be convenient to produce green preserved peas without the use of copper, it was by no means necessary that peas should be green in order to be eaten[1] (*vide* also under "Food Poisoning.").

Charteris and Snodgrass show that by digesting coppered peas in dilute hydrochloric acid some copper is dissolved. By digesting the peas with a mixture of pepsin and dilute hydrochloric acid, copper was dissolved to the extent of 0·52 grain per pound of peas, equal to 2·05 grains of sulphate of copper. Artificially prepared compounds of copper with albumen and with casein were also submitted to the action of solutions containing respectively pepsin with hydrochloric acid and pancreatin with carbonate of sodium, the temperature being maintained at 99° Fahr. It is stated that at the end of an hour and ten minutes digestion was complete in the acid solution in the case of the albumen compound, and at the end of two hours in the alkaline solution ; in the case of the casein compound, digestion was complete at the end of two hours in the acid solution, and at the end of one hour in the alkaline solution. Rabbits and pigs were supplied with food containing an admixture of albuminate of copper or sulphate of copper, and when the animals were killed copper was found in their liver and kidneys. When the quantity of sulphate of copper given to a young pig weighing nine pounds amounted to 60 grains, it refused the food for twenty-four hours, but it readily took food containing 10 grains, and continued to do so for four days, when it was killed.

Drummond[2] states that about 50 mg. of copper per kilogramme is given as the usual amount in greening vegetables. A certain amount of this copper is liberated in a dialysable form, and therefore able to be absorbed into the body. He found copper in the liver of rats fed on greened peas, but could find no evidence of pathological change in the organs, nor did there appear to be any difference in the health of animals so fed as compared with the control animals. No details are given of the tests.

Toxicity and Fatal Dose. The ordinary pharmacopœial dose of the sulphate is a quarter to two grains, for emetic purposes five to ten grains.

There are but few instances in which the sulphate has proved fatal in the human subject, so that but little is known of the fatal dose. A girl, sixteen months old, put some pieces of *Bluestone* (sulphate of copper), which was given to her to play with, into her mouth. In a quarter of an hour the child vomited a bluish-green coloured matter, with pieces of sulphate of copper in it ; the skin was alternately cold and hot, but there was no purging. The child died in *four hours*, without being convulsed, but it was insensible before death.[3] Unfortunately no inspection of the body was made.

A woman who swallowed *two ounces* of verdigris died in three days : in addition to the symptoms described below, there were convulsions and paralysis before death. Niemann relates that a female, aged twenty-four, swallowed *half an ounce* of verdigris, and died with symptoms of severe irritation of the stomach in sixty hours.[4] In consequence of the great uncertainty of its operation, subacetate of copper is not employed internally.

[1] *Pharm. Journ.*, November 7th, 1891, p. 366.
[2] *The Analyst*, October 1925.
[3] *Med. Gaz.*, vol. 18, p. 742.
[4] "Taschenbuch," p. 458.

Duration. As with all irritants, the symptoms appear within a quarter of an hour, or less, of taking the poison ; but exceptionally, owing to the condition of the stomach contents, they may not appear for three or four hours.

Symptoms. The sulphate speedily causes violent vomiting, for which purpose it is sometimes used. If the dose has been excessive, in addition to the vomiting, there is headache, and pain in the abdomen, with purging ; the pain is of a colicky character ; in aggravated cases there are spasms of the extremities and convulsions. Paralysis, insensibility, and even tetanus, have preceded death, when the poison was administered to animals. Among the symptoms occasionally met with in the human subject may be mentioned jaundice. This has been observed to attend poisoning by the sulphate, as well as by Scheele's Green.

Verdigris produces symptoms somewhat similar to those caused by the sulphate. There is a strong styptic metallic taste, with a sense of constriction in the throat, followed by severe colicky pains, vomiting of a green-coloured liquid, and purging, with violent straining (tenesmus).

A peculiar case of copper poisoning is reported by Gardner.[1] Some fine particles of bronze from a bearing contaminated the flour from which bread was made. During fermentation a certain amount of acetic acid was formed, which reacted with the copper to produce verdigris. Symptoms of vomiting, diarrhœa and pyrexia were observed in those who ate the bread.

Chronic poisoning by copper is occasionally seen among workers in this metal and its salts. The poison enters the system by absorption from the alimentary canal, by the lungs in the form of dust, and partly by the skin in handling the metal or its salts. The usual symptoms are a coppery taste in the mouth, giddiness, pain in the bowels, vomiting, occasional diarrhœa, and wasting of the body. A green line on the margin of the gums may occasionally be seen.

The existence of chronic copper poisoning appears to be accepted by the majority of toxicologists ; but it must be admitted that much has been alleged as to this form of disease which is open to question. The fact that the majority of workers in copper and its compounds remain perfectly healthy is, however, not conclusive of the innocuous character of the metal.

The disease known as brass-founders' ague or metal fume fever[2] is found in workmen engaged in smelting brass which is an alloy of copper and zinc, is due to the inhalation of the fumes of these metals and probably mainly due to the zinc or impurities, such as cadmium.[3]

Treatment. This can be on general principles only, *vide* p. 250.

Post-mortem Appearances. In poisoning with the salts of copper, the mucous membrane of the stomach and intestines has been found more or less thickened and inflamed, in the few fatal cases which have been hitherto examined ; the membrane has also been found destroyed and softened in poisoning with verdigris. The gullet has presented an inflamed appearance. In a case of poisoning with verdigris quoted by Orfila, the stomach

[1] *B.M.J.*, October 31st, 1925.
[2] *Vide B.M.J.*, 1900, 1, in an article by Dr. Wm. Murray, also *B.M.J.*, 1901, 2, p. 405
[3] Dronker and others, *Journ Indus. Hyg.*, 9 : 187 (1927).

was inflamed and thickened. The small intestines were inflamed and perforation had taken place. The rectum was ulcerated on its inner surface.

Analysis. The *vomited* matters are generally of a *blue* or *green* colour ; and broken crystals of the blue vitriol may be discovered in them. If the green colour of the vomited liquids is due to altered bile, it will not acquire a blue tint on adding to a portion of the green liquid a solution of ammonia ; but if it be caused by a salt of copper, this change of colour will serve to indicate the fact.

The salts of copper are generally known by their colour ; whether in the solid state or in solution, they are either blue or green. Nickel and some ferrous salts are also green ; but there are very marked chemical differences between the salts of these metals and those of copper. There are *three* very *soluble* salts of copper ; two of these are blue, the sulphate and nitrate, and one green, the chloride. The solutions of the copper salts have generally an acid reaction. The salt should be dissolved in water, diluted, and the following tests may then be applied :—

Tests. (1) *Solution of ammonia* gives a bluish white precipitate, which is soluble in an excess of the reagent, forming a deep violet-blue liquid. Nickel salts also give the deep blue colour with ammonia, but differ from copper salts in their response to the other tests. (2) *Ferrocyanide of potassium* gives a rich claret-red precipitate ; if the quantity of copper is small, the liquid acquires merely a light red colour. This is an extremely sensitive test for copper, and will show the presence of one part per million of copper. It may be used as a quantitative test. If the solution contains copper in quantity, the precipitate is of a deep red-brown colour, and of a gelatinous consistency. Ferrocyanide of potassium will act on the violet-blue solution produced by ammonia, provided acetic acid is added to neutralise the ammonia. One portion of the liquid may thus be tried by the two tests. Iron, present in addition to copper, masks the red colour by the intense blue which it produces itself with ferro-cyanide. (3) *Sulphuretted hydrogen gas*, or sulphide of ammonium, gives an almost black precipitate even in an acid solution, or, if the copper is in low concentration, merely a reddish-brown colour. (4) A slip of *polished iron* (a common needle), suspended by a thread in the liquid, slightly acidified with sulphuric acid, is speedily coated with a layer of copper, even when the salt is present in a very small proportion. If the needle is left for some days in the liquid, the iron will be slowly removed, and a hollow cylinder of metallic copper will remain. This may be washed, dissolved in dilute nitric acid, and tested with the foregoing tests ; or the iron, coated with copper, may be at once partially immersed in ammonia and exposed to air. The liquid then becomes slowly blue. Half a grain of sulphate of copper, dissolved in sixteen ounces of water, may thus be easily detected. The blue solution, acidified with acetic acid, gives a red colour or precipitate with ferrocyanide of potassium. (5) *The Electrolytic Test*. If a few drops of the copper solution are placed on platinum foil, slightly acidified with a diluted acid, and the platinum is then touched through the solution with a slip of zinc, metallic copper, recognisable by its colour, is immediately deposited on the platinum. When the quantity of copper is small, there is merely a brown stain ; but a blue liquid is formed by

pouring on it ammonia, and exposing it to air. The blue liquid may be further tested with ferrocyanide of potassium after acidification with acetic acid.

Commercial sulphate of copper sometimes contains *traces of arsenic*. Ten grains of the crystallised sulphate will occasionally be sufficient to yield evidence of the presence of arsenic. When the sulphate has been given as an emetic, traces of arsenic may sometimes be found in the contents of the stomach or in the matters vomited.

Copper in Organic Liquids. The copper is liable to be precipitated by certain organic substances, *e.g.*, albumen, fibrin, casein, and mucous membrane; but some of these organic compounds are easily dissolved by acids, or even by an excess of the solution of copper salt. A portion at least of the salt of copper is, therefore, commonly held dissolved. In such cases there is one peculiar character possessed by these liquids; *i.e.*, they have a decidedly *green colour*, even when the copper salt is in a far less than poisonous proportion.

In the Tissues or Urine, or in Vegetable Matter. Dry and incinerate the organic matter, using an iron burner, digest the residue in warm nitric acid, and then evaporate to dryness. The residue may be dissolved in a small quantity of water, and a polished needle immersed in it for some hours. The metallic deposit, if any, on the needle may then be recognised as copper by its colour and by the tests already described.

In very small amounts copper is present in the liver of man. Dupré found in human livers about one part of copper in 500,000; and in fourteen bodies examined by Bergeron and Hote copper in minute quantities was found in every case. It also occurs normally in very small quantities in the kidneys and in the blood. Indeed, it is now known that, though needed only in very small amounts, copper is an essential constituent of most living organisms. The copper is probably mainly derived from the brans of cereals, such as wheat and barley; in turnips, and most other vegetables, copper in very minute traces may generally be detected. It has been suggested by Johnstone that the presence of copper in cereals may be due to the practice of dressing the grain, and also the ground, with sulphate of copper, with the object of protecting it from the ravages of vermin after the grain is sown (Luff).

Cases. The following case has a certain interest :—

"A farm labourer was working on a farm on August 19th and 20th. He had a spraying machine strapped on his back and was spraying potatoes. On the 19th he complained before dinner of being sick; on the 20th he went home, partook of some supper (potatoes) at seven o'clock, and retired to rest between nine and ten o'clock. In the early morning he was taken severely ill, and at 4.30 a.m. on the morning of the 21st suffered from severe cramps, failing heart, and free perspiration. He died at about two o'clock in the afternoon of the 21st. He had a fresh wound on his hand, which was perfectly blue with 'bluestone,' which was chiefly sulphate of copper. Medical evidence was given that deceased had died from collapse following irritant poisoning from absorption from the hands and from breathing in the fine vapour of the spray. The jury recorded that the deceased died 'from collapse following upon irritant poison,' possibly through the absorption of the mixture sulphate of copper used in the spraying machine which he carried and used on August 19th and 20th."

In 1866 a remarkable set of cases occurred in a family at Itchin Abbas, Hants, in which twelve or more members of the family suffered from symptoms of poisoning similar to those produced by copper. A badly tinned copper vessel had been used for cooking the food, with much salt. One patient, an old man, *æt.* 90, died after

three weeks, the others recovered. A set of cases is reported to have occurred at Geneva, in 1870, in which ten persons were taken ill with symptoms of irritant poisoning, and four died. It was found that the food had been cooked in a copper utensil containing a large quantity of verdigris.[1]

Poisoning by Gold Salts

Source. The *Trichloride* is a powerful irritant poison, acting locally like nitrate of silver.

Gold salts were at one time used in the treatment of syphilis. Because of the bactericidal action of gold on the tubercle bacillus, it has been used in the treatment of tuberculosis but this practice has fallen into disuse because of the difficulty in obtaining safely an effective concentration. Nowadays gold salts are not greatly used in medicine, though they are sometimes effective in lupus erythematosus, and favourable results have been reported in rheumatoid arthritis. The salt most often employed is sodium gold thiosulphate. Toxic reactions are common—headache, giddiness, pulmonary œdema, cardiovascular collapse, toxic nephritis, acute hepatitis, severe skin reactions, thrombocytopenia, agranulocytosis, aplastic anæmia have all been recorded. Fatal reactions are said to occur in 1—3 per cent. of cases.[2]

The use of B.A.L. (2 : 3 di-mercapto propanol) in cases of poisoning by gold salts is referred to in the section on arsenic poisoning (see p. 418).

Case. In October, 1893, a case of poisoning by chloride of gold was admitted to Guy's Hospital, of which the following is an abstract :—

A boy of six swallowed the contents of a small glass tube found on a dustheap. He was sick immediately afterwards, the first vomit being black in colour. The vomiting persisted, and he was admitted to hospital at 2 a.m., much collapsed, eyes closed, face pallid and cold, the lips, tongue, teeth, stained a purplish colour. The fauces were injected, but not stained ; the fingers were stained. Pulse extremely feeble. Within ten minutes the bowels acted twice. At 10 a.m. the vomiting still continued, and diarrhœa was present. He did not seem to be in pain, but the epigastrium was tender on pressure. He had by this time rallied considerably from shock. There was noticed a black stain on the teeth. About 5 p.m. on the day of admission the vomiting and diarrhœa ceased, and he was discharged well about twelve days after admission. Pupils natural and no unconsciousness throughout.

Sir Thomas Stevenson reported that the tube was one used by photographers for toning ; it was made to contain 15 grains. Of this 2·6 grains were found in the tube, leaving 12.4 grains either swallowed or lost. He found traces of gold in the fæces and vomit, but none in the urine.

Poisoning by Preparations of Thallium

The salts of this metal are highly poisonous, and have frequently caused severe toxic symptoms when used in therapeutic doses. The principal toxic phenomena are stomatitis, diarrhœa, dyspnœa and pain in the extremities.[3]

The salts are soluble, colourless, and nearly tasteless ; and therefore may be easily administered and have been found to operate through the skin by absorption.

In 1898, Sabouraud[4] used thallium acetate as a depilatory in the treatment of ringworm, but abandoned it on account of its toxic properties.

[1] *Lancet*, 2 : 622 (1901).
[2] *Lancet*, 1937 : i, 554 ; ii, 784, 834.
[3] *Buschke, Deutsche Med. Woch.*, 1910, 37, 161.
[4] " Entretiens Dermatologiques," Paris, 1913.

Its use was periodically revived, but was not widespread until Buschke and Peiser,[1] in 1922, recommended it to be given by mouth in a dose of 8 mg. per kilo body weight. Since then, thallium acetate has been widely used in the treatment of ringworm in children, the usual method consisting in the administration of a single dose of 8·5 mg. per kilo body weight. Epilation is usually complete seventeen to twenty-two days later, but has been observed as early as the fifth day. It is generally agreed that slight toxic symptoms are to be expected in about 40 per cent. of the cases—muscular pains in the legs of a pseudo-rheumatic character, lethargy, slight headache, and, sometimes, erythematous rashes—but generally disappear within three weeks. It has been stated[2] and denied[3] that these symptoms are commoner in older children

At least twenty-four deaths have been due to the use of thallium acetate in the treatment of ringworm. According to Ingram,[4] however, all must be ascribed to faulty technique, and in three of them at least[5] the fault lay in a dispenser's error, which led to a considerable overdosage. In 1930, fourteen school-children died in Granada during treatment for ringworm.[6]

Thallium acetate, in doses of 8 mg. per kilo of body weight, was administered to *sixteen children* suffering from ringworm. All but two of them subsequently died. The first death occurred two days after administration ; there were four deaths on the fifth day, three on the sixth, one on the seventh, two on the eighth, one on the tenth, one on the eleventh, and one on the sixteenth. Symptoms of poisoning are described as follows : Digestive system : acute abdominal pain, glossitis, no constipation or true diarrhœa, vomiting of cerebral type after the fifth day. Cardiovascular and respiratory systems : acceleration of pulse rate only after a few days, respiratory difficulty in some cases, one death in forty-eight hours from bronchopneumonia. Urinary system : decreasing albuminuria in five cases dating from onset of illness, progressive oliguria, and in three cases anuria. Nervous system : profound, early, and persistent *somnolence*, with occasional evidence of excitement, supraorbital and *frontal headache*, *joint pains* from twelve hours after ingestion onwards, *muscular clonus* in half the cases, persistent tic de salaam in all ; loss of corneal reflex (and, in one case, of light reflex), ptosis in six cases, and conjunctival congestion in the majority. Other cranial nerve symptoms included mydriasis, miosis, anisocoria, dim vision, rotatory nystagmus. Other symptoms were pain in the limbs, slight rise of temperature, and cyanosis of extremities and of face. One of the oddest features of the outbreak of poisoning was the delay in depilation—usually expected to be marked between the fifteenth and twenty-second days after administration. In this series none of the fatal cases showed any sign of depilation at all, and in the *two survivors the process began as late as the twenty-ninth day.*

Post-mortem, Leche Marzo's sign was positive in thirteen cases. There was engorgement of cerebral sinuses and meningeal vessels, *subpleural hœmorrhage* and congestion of trachea and bronchi, *punctate effusions and ecchymoses in the pyloric antrum*. Histological examination revealed nothing abnormal in the pons or medulla beyond an increase in the number of nuclei.

Mahoney,[7] in a paper which reviews the literature of thallium poisoning, records three cases of retrobulbar neuritis following the use of a thallium-containing depilatory cream. In one case there was also

[1] *Derm. Woch.*, 74 : 443 (1922).
[2] Urueña, " Le Traitement des Taignes par l'acétate de Thallium," Paris, 1928.
[3] Percival, *Brit. J. Derm. Syph.*, 42 : 59 (1930) ; Lewis and Lloyd, *B.M.J.*, 2 :99 (1933).
[4] Ingram, *B.M.J.*, 1 : 8 (1932).
[5] Lynch and Scovell, *Lancet*, 2 : 1340 (1930).
[6] *Brit. Med. Journ.*, 1 : 26 (1924).
[7] *J. Amer. Med. Assoc.*, 1932, February 20th, 618. See also *Ibid.*, Munch, 1934, *192*. 1929.

bilateral motor and sensory involvement of the legs. Rapid and continuous improvement of vision took place when the patients stopped using the cream.

Thallium is widely used as a rat poison (*e.g.*, Zelio-paste, which contains 2 per cent. of thallium) and in this form has caused a number of cases of acute poisoning, many of which were fatal—suicide, accident, and murder. The following case is quoted by Leschke[1] :—

> In Vienna, in 1927, a woman murdered her husband by mixing Zelio-paste with his food (three tubes). He vomited, and had diarrhœa with violent colic, and the diarrhœa was succeeded by constipation. Later there was acroparæsthesia with decreased tactile sensitivity but increased sensitivity to touch, muscle and joint pains, and increasing dimness of vision. He became completely bald, and suffered from acute blepharo-conjunctivitis and purulent dermatitis of the face. Terminally, he was completely demented. The *post-mortem* examination showed swelling of the brain, with degenerative changes in the liver, kidneys and heart.

Lewis and Lloyd[2] call attention to the fact that the toxic manifestations of thallium are markedly similar to those of lead (which it also resembles in being a cumulative protoplasmic poison). Both metals have a preference for nervous tissue, and thallium poisoning, like lead poisoning, frequently gives rise to cramp-like abdominal pains, peripheral neuritis, and blurred vision. A blue line on the gums has also been observed in thallium poisoning.

Survival of the acute poisoning may be followed by nephritis, paralysis, blindness, and mental disorders.

Chronic thallium poisoning may occur in workers engaged in the manufacture of rat and vermin poisons, depilatory pastes, luminous paints, and in the glass and dye industries. Besides falling out of the hair, inhibition of growth and of sexual development, nephritis, degeneration of the cardiac muscle, peripheral neuritis, and mental disturbances have all been described. Böhmer[3] reports changes in the finger-nails similar to those occurring in arsenic poisoning.

Post-mortem Appearances. These depend on the length of time which has elapsed between the ingestion of the poison and death. There may be slight gastric irritation with sub-mucous petechial hæmorrhages, fatty degeneration of the heart, congestion and fatty degeneration of the liver, cerebral congestion, subpleural hæmorrhages and congestion of the trachea and bronchi. The kidneys may show congestion, swelling of the glomeruli, cloudy swelling and necrosis of the cells of the convoluted tubules.

Treatment. Little is known of specific treatment of thallium poisoning. Leschke recommends intravenous injection of 20 c.c. daily of a 3 per cent. solution of sodium thiosulphate. Probably the safest course is to follow the general procedure in heavy metal poisoning.

Analysis. The following procedure is recommended by Roche Lynch (*Lancet*, 1930, 2, 1340).

To the filtrate from the Fresenius process, add ammonium chloride and ammonia until the liquid is alkaline, and boil. Filter off and discard

[1] "Clinical Toxicology," 1934, p. 31 : also *B.M.J.*, February 21st, 1931.
[2] *B.M.J.*, 1933, July 15th 99.
[3] Böhmer, K., *Deut. Zeitsch. f. ger. med.*, 30 : 270 (1939).

the precipitate (iron, calcium, magnesium, chiefly as phosphates). To ensure complete removal of phosphate it is well to add calcium chloride before the ammonium chloride.

Saturate the filtrate with hydrogen sulphide, or add excess of freshly prepared ammonium sulphide solution. Filter off the dark precipitate, and wash it thoroughly, first with ammonium sulphide solution and finally with distilled water. It consists of thallous sulphide and sulphides of other Group II metals (copper is the only probable one, in toxicological analyses). Dissolve the precipitate in hot dilute hydrochloric acid (copper sulphide remains undissolved), make the solution alkaline with ammonia, and boil. There should be no precipitate if separation has been complete, but any precipitate which does form is to be filtered off and discarded. Make the solution faintly acid with hydrochloric acid and add excess of potassium iodide solution. Thallium gives an immediate yellow precipitate of thallous iodide. Boil and allow to stand for twelve hours before filtering, since the precipitate may be colloidal at first. Filter and collect the precipitate in a Gooch crucible, wash first with potassium iodide solution and then with alcohol until the washings are free from iodide, dry at 120° C. and weigh. (Thallous iodide is slightly soluble in water (1/17,000), but practically insoluble in potassium iodide solution or in alcohol.)

The identity of the precipitate may be confirmed by

(1) its difficult solubility in sodium thiosulphate solution, in which yellow lead iodide is easily soluble.

(2) the characteristic green line in the spectrum obtained when it is burned in the Bunsen flame on a platinum wire.

REFERENCE

Buschke and Peiser. "Ergeb. der. allgem. Path.," 1931, 25 (with full bibliography).

Poisoning by Salts of Tin

Tin salts are not particularly common poisons and very few deaths have been attributed to their use. The tetrachloride is used in weighting silk but with the information available it would be difficult to ascribe any chronic toxic effect from its use in that industry.[1]

Tin salts may be found in the form of whitish yellow crystals or more commonly in acid solution. They cause gastro-intestinal irritation, pain, vomiting, diarrhœa, cyanosis and collapse. The organic compounds of tin, tin methyl and tin ethyl may cause neuritis, including retinal neuritis.

Luff has described four cases of tin poisoning caused by tinned cherries. The syrup of the cherries was strongly acid, the acidity being mainly due to malic acid ; the juice contained tin in solution equal to 1·9 grain of the higher oxide of tin in each fluid ounce, which would be equal to 3·2 grains of the malate of tin in the same quantity of juice. The symptoms of these cases were very severe, there being a considerable amount of collapse and cyanosis and in one case unconsciousness : it was estimated that the symptoms were produced by doses of the malate of tin varying from four to ten grains. All the cases recovered.[2]

[1] *Jour. Ind. Hyg.*, 6 : 28 (1924).
[2] *B.M.J.*, 1 : 833 (1890).

Treatment. The stomach should be washed out with a solution of bicarbonate of soda in warm water and in general treat as for heavy metals.

Analysis. Tin may be separated from organic admixture by destroying organic matter by the moist method (see p. 442). The clear fluid, which contains *stannic* chloride, may be submitted to the following tests:—

(1) Sulphuretted hydrogen gives with a solution of a stannous salt a brown precipitate of stannous sulphide, and with a solution of a stannic salt a yellow precipitate of stannic sulphide.

(2) Stannous chloride added to a solution of mercuric chloride first reduces it to the mercurous state, throwing down a white precipitate of calomel, and subsequently reduces this to the metallic state, producing a grey precipitate, due to the formation of finely divided mercury.

(3) With gold chloride stannous chloride gives a purple precipitate (*vide* also tests for zinc).

(4) With sodium hydroxide solution, stannous salts give a white precipitate soluble in excess of the alkali, from which solution black tin oxide is precipitated on boiling. Stannic salts similarly form a white precipitate with sodium hydroxide, soluble in excess of alkali, but not reprecipitated on boiling.

From the solution of stannic chloride the metal is deposited on zinc by zinc-platinum couple, and on dissolving the tin deposit in boiling HCl a solution of stannous chloride is obtained suitable for the tests quoted.

Poisoning by Lead and its Salts

Lead poisoning, or plumbism, has been known since ancient times, and is the subject of an enormous literature. Its history has been discussed by Teleky.[1] As with other metals, the compounds of lead are more dangerous than lead itself, except when lead is in the volatile state, and generally, the more soluble the compound, the more poisonous it is. Lead may obtain access to the body by inhalation, by ingestion, or by absorption from the skin or mucous surfaces. It has been stated that lead is ten times more dangerous when inhaled than when ingested.[2]

All compounds of lead are liable to produce toxic effects, including the acetate (sugar of lead), the oxycarbonate (white lead), the oleate as diachylon plaster, the oxides including yellow and red lead, the chromate and the organic compounds.

The toxicity of lead compounds is influenced by many factors, such as solubility in body fluids—which is not necessarily the same as their solubility in water—the length of time they are in contact with the body fluids, the quantity ingested or inhaled or absorbed, and the amount actually in the circulation. Lead stored in the bones is not dangerous until it passes into the circulation in amounts too great to be eliminated.[3]

Lead may produce acute symptoms of gastro-intestinal irritation, but its importance in toxicology is due mainly to chronic poisoning in industry by the ingestion of repeated small doses.

Chronic lead poisoning may occur in workers in lead: in mining and milling lead ores such as the carbonate, oxide and sulphide: in the paint industry from sulphates, carbonates, oxides and chromates: in the

[1] Teleky, *Ind. Med.*, Ind. Hyg. Section, 9 : 17 (1940).
[2] Drinker, *Ind. Med.*, 4 : 253 (1935).
[3] Mayers and McMahon, *N. Y. State Dept. Labour Spec. Bull.*, 195 (1938).

pottery industry from lead silicate ; and from the peroxide, red lead and litharge in the making of storage batteries. Lead acetate, nitrate, chloride and tetra-ethyl lead are also met with in industry.

Subacute or chronic lead poisoning may be caused by the contamination of food or drink. Details of all such hazards are available in any book on industrial poisoning.[1, 2, 3, 4, 5]

Altogether, 1,250 cases of industrial lead poisoning were notified in 1899 ; by 1938 the number had fallen to less than 100. The figures show the improvement in the national health and care in dangerous occupations. The incidence of lead poisoning is not exactly known, however, as no doubt many cases escape detection. Since 1895 poisoning by lead, phosphorus, and arsenic, contracted in a factory or workshop has been notifiable in Great Britain, where between 1900–1909 there were 6,762 cases with 245 deaths. These figures, apparently small, only apply to cases in factories and workshops, and do not include those in plumbers and painters. In connection with these two occupations there were 239 cases with 44 deaths in 1908, and 241 cases with 47 deaths in 1909 (quoted by Legge).

1. **Metallic Lead.** Metallic lead in itself may be dangerous when ingested but becomes much more toxic when inhaled in the form of lead fumes In most of the cases to be mentioned it is ingested as an oxide or carbonate or some other salt ; but there are cases of poisoning by the metal itself, which has in the body undergone a change into a salt of lead.

Except in acute irritant cases, it seems to be the lead which is the poisonous constituent.

A tea-dealer was seized with symptoms of lead poisoning, and the cause remained long unsuspected, until he admitted that, in course of his trade, he had the idle habit of often placing pieces of tea-lead in his mouth, and crushing the metal between his teeth.

Sonnenkalb considered that snuff frequently acquires an impregnation of lead on account of the lead coverings in which it is packed. He collected nineteen cases of this form of chronic poisoning ; in fourteen of these there was paralysis, and in five there were symptoms of gastric disturbance. The arms were most commonly affected, with paralysis and wasting of the extensor muscles. In twelve cases there was a blue colour of the gums. All suffered from colicky pains and constipation. The poisoned snuff had been used for a period of from six months to twenty years : and on leaving it off the patients improved rapidly, and eventually recovered. Snuff had actually been adulterated with red lead to improve its colour.

Cases have been recorded of chronic lead poisoning caused by lead shot, shrapnel bullets, etc., which have remained embedded in the tissues. The symptoms may appear within a few weeks or only after years.

Lead poisoning in babies may result from the use of lead nipple-shields.[6]

[1] Local Government Board Med. Dep. "On Lead Poisoning and Water Supplies," Lond. 1903.
[2] Hamilton, A., "Industrial Poisons in U.S.A."
[3] Aub, Fairhall, Minot and Reznikoff, "Lead Poisoning" (1926), Baltimore.
[4] Legge and Goadby, "Lead Poisoning and Lead Absorption," London, 1921.
[5] Hunter, Donald, "Industrial Toxicology," Clarendon Press, Oxford, 1944.
[6] *Jour. A. M. A.*, 86 : 1514 (1926).

2. **Sugar of Lead or Lead Acetate.** This is more frequently taken as a poison than any of the other salts of the metal, although cases of acute poisoning by lead in any form are not common. The substance is met with in solid heavy crystalline masses, white or of a brownish white colour ; it resembles loaf sugar in appearance, and has often been mistaken for it. It has also a sweet, followed by an astringent or metallic taste. It is very soluble in water, four parts of distilled water at 60° Fahr. dissolving one part ; and it is much more soluble at a boiling temperature.

A remarkable series of cases of poisoning by acetate of lead has been reported.[1] By some accident, about thirty pounds of this substance were mixed at a miller's with eighty sacks of flour, and the whole was made into bread by the bakers and supplied as usual to their customers. It seems that no fewer than 500 persons were attacked with symptoms of poisoning after partaking of this bread.

Liquids used for culinary or dietetic purposes, especially if they contain a free *acid*, are liable to become impregnated with lead, derived from the glaze of the vessel in which they are kept, and thus form poisonous salts. If vinegar is used, acetate of lead may result. Litharge-glaze is also easily dissolved by alkaline or *fatty* substances. The eating of dripping, or the fat of meat baked in a newly glazed vessel, has been known to give rise to a slight attack of colic ; while the symptoms were referred by the person to some substance mixed with the food. In 1852, four men partook of rhubarb-pie and milk for supper. Shortly afterwards they were all seized with violent vomiting and intense colic. Lead was detected in a portion of the vomited matters and food. The only source to which the lead could be traced was the glaze of the pans in which the milk was kept.

The acetate, in solution as liquor plumbi subacetatis, is used in medicine as a lotion, and from this solution as a basis many proprietary hair-dyes, washes, etc., are made. Of such, Goulard extract or water is an example, which has caused death in at least four instances, one in France and three in England.

A case is recorded of a woman who took lead acetate, dissolved in water, three times a day for a month in an effort to procure an abortion, ingesting in all 110 grains of the salt. She suffered from fairly severe symptoms of lead poisoning but recovered after treatment.[2]

A case of severe plumbism from washing out the nose with a lotion containing " a pennyworth of sugar of lead to the half-pint of water " has been reported. This washing had been done two or three times a day for some months.[3]

3. **White Lead.** This salt is responsible for the majority of cases of lead poisoning occurring amongst workmen, inasmuch as it is the basis from which many lead paints and glazes are made.

One cause of lead-palsy among infants may be the use of farinaceous food wrapped in lead-foil having a thinly tinned surface, sold as patent tin-foil. Such infants' food is sometimes strongly impregnated on the outside with carbonate of lead. Snuff and tobacco, chocolate, and other substances in ordinary use are frequently wrapped in this spurious tin-foil. If the articles are kept in a damp place, they may thus become impregnated with carbonate of lead.

[1] *Lancet*, 1849, 1, p. 478.
[2] Tompsett, S. L., *Lancet*, 1 : 994 (1938).
[3] *B.M.J.*, 1 : 491 (1898).

White lead is sometimes contained in small proportions in loaf sugar, owing to the moulds in which the sugar is set being painted with white lead, and a portion being thus mechanically taken up.

Lacy has pointed out the injury to health which is likely to follow the use of white lead as a cosmetic by actors. The glazed white leather lining of hats is strongly impregnated with carbonate of lead, which may penetrate the body through the perspiring skin.

In 1881, a little girl, aged two years and a half, was noticed to be feverish and restless, and was reported to have had a bad night. The following day she vomited constantly, was unable to take any food, and suffered greatly from thirst. It was considered that she was suffering from severe gastric catarrh, induced probably by sucking cheap coloured crayons, the pink mark of one of which was noticed on the child's mouth. In a few days the child rallied, and it was thought that all danger was over; but on the twenty-third day of the illness she was seized with convulsions, and the next day she died. All the organs were healthy, with the exception of the brain and stomach. The left side of the brain is reported to have been distended with fluid, whilst the stomach presented signs of acute inflammation, and in two places perforation had occurred. Either the effusion into the ventricles or the condition of the stomach might, it was considered, have accounted for the fatal issue. An analysis of the crayons showed that they all contained poison. The most poisonous was a pink one, which contained more than half its weight of white lead, coloured with an innocuous vegetable substance. The weight of this crayon was fifty grains. Another crayon contained Prussian blue, mixed with Dutch pink.[1]

4. **Oxides of Lead.** The yellow oxide (massicot) and the puce-coloured oxide (peroxide) are but little known, except to chemists. *Litharge* and minium or *red lead* are, however, much employed in the arts, and have sometimes given rise to accidental poisoning.

A woman who had swallowed two and a quarter ounces of red lead was admitted into hospital. No symptoms appeared for nine hours. There was then colicky pain, with urgent vomiting, followed by headache and general tenderness of the abdomen. She entirely recovered in about twelve days.[2] In March 1870, owing to an accident, some red lead became mixed with a quantity of beer at a brewery at Guildford. Several persons who drank this beer suffered from lead poisoning. One man died, but it was probable that disease of the lungs was the immediate cause of death. Colicky pains, a blue line on the gums, and constipation, were well-marked symptoms.

5. **Other Salts.** This group is meant to include all those cases in which the lead gains access to the body by what may be called domestic or trade routes. It includes some of the above salts, undoubtedly; but often, and especially in water and beer, it may be doubtful what special salt is present.

Cosmetics and hair-dyes containing preparations of lead, commonly called hair-restorers, may also produce dangerous effects. The author met with an instance in which paralysis of the muscles on one side of the neck arose from the imprudent use of hair-dye containing litharge. These hair-dyes or "hair-restorers" are sometimes solutions of acetate of lead of varying strength in perfumed and coloured water. In other cases they consist of hyposulphite of lead dissolved in an excess of hyposulphite of sodium. In one instance the continued use of such a dye is reported to have proved fatal, and lead was found in the liver and in one of the kidneys.[3]

[1] *B.M.J.*, 1882, I, p. 669.
[2] Guy's Hosp. Rep., 1850, p. 209.
[3] *Pharm. Jour.*, 1869, 1, p. 304; also January, 1869, p. 440.

Bagchi has described toxic effects in Hindu women and children from the use of a red vermilion hair-dye.[1]

An instance of the fatal effects of cider contaminated with lead is reported to have occurred in Worcestershire in 1864, and another fatal case occurred in Herefordshire in 1867. Eight men were seized with symptoms of lead poisoning, and one died. Herapath found one grain of lead in a gallon of water. The leaden pipe was found corroded by the acid in the cider.

Lead pipes are largely used by publicans for the supply of beer. It is possible, therefore, if the beer is acid, and is allowed to remain some time in the pipe, that the first portions drawn may acquire an impregnation of lead, which may give rise to colic and other unpleasant symptoms. This is a not infrequent and often unsuspected cause of lead poisoning in potmen, though by them sometimes attributed to handling and cleaning pewter vessels.[2]

Litharge was formerly much used to remove the acidity of sour *wine*, and to convey a sweet taste. Acetate of lead, or some other vegetable salt of the metal, is in these cases formed ; and the use of such wine may be productive of alarming symptoms. Many years ago, a fatal epidemic colic prevailed in Paris owing to this cause ; the adulteration was discovered by Fourcroy, and it was immediately suppressed. Wine thus poisoned is known by its being blackened by sulphuretted hydrogen.

With regard to water, Taylor wrote in previous editions as follows : " Water which contains more than one-twentieth of a grain of lead per gallon should be rejected as unsafe. Such a water gives a sensible darkening of colour when a wineglassful of it is placed in a white porcelain dish, acidulated with a few drops of hydrochloric acid, and then a drop of sulphide of ammonium added. Soft waters, more especially those moorland and ferruginous which are acid, and those contaminated with the products of decomposing sewage, are especially prone to act upon metallic lead, and where delivered at house taps often contain half a grain or more of lead per gallon."

Such a limit is far too high. All lead contamination is objectionable, and no degree of it can be considered safe. Lead is an accumulative poison, and it is not so much the quantity taken in which determines the symptoms of lead poisoning as its continued introduction.

An infant was paralysed by reason of its having been washed every morning with water containing a finely diffused oxycarbonate of lead (A. S. Taylor).

Sleeping or working in newly painted rooms is another source of plumbism.

The author suffered from a severe attack of colic as a result of sitting in a room for a few hours a day in which a large surface of canvas for an oil-painting had been covered with white lead and drying oil. An officer, *æt.* 50, fond of painting in oil-colours, worked for some time in a room eight feet square which had a large stove in it. He was attacked with wristdrop (paralysis) and soon afterwards with paralysis in both legs. It appears that his servant always ground his colours, mixed them, and cleaned his brushes. The officer had experienced an attack some years before ; but from this, by laying aside oil-painting, he completely recovered. The symptoms were of the character peculiar to lead poisoning, and as they disappeared on the removal of the patient to another atmosphere, there could be no doubt about the case.

[1] *Ind. Med. Gaz.*, 76 : 23 (1941).
[2] *Vide Lancet*, 1860, 1, 60.

These insidious effects of lead should be borne in mind by those who deny that any noxious emanations can escape from papers in inhabited rooms merely because the majority of persons who live in them do not suffer.

A case of lead poisoning was traced to the handling of vulcanised rubber impregnated with lead. The man was a trunk-maker, and used this material in his trade.[1] The mere handling of lead or its compounds is therefore sufficient to produce all the effects of chronic poisoning.

The above brief sketch of the modes of occurrence leads practically to the conclusion that lead can enter the body by any route and in as many disguises as we can imagine.

The occurrence and prevention of lead poisoning from water and from trade processes is a province of hygiene, and cannot be discussed here. Plumbism is now a trade disease under the Workmen's Compensation Act (*vide* Vol. I).

6. **Tetra-ethyl Lead.** This substance has been introduced for use with motor fuels to increase their efficiency. Within 17 months of its introduction in 1923, eleven deaths were reported among the workmen making it, and in 1923, 149 cases of encephalopathy occurred in three factories,[2] so that tetra-ethyl lead must be looked upon as a dangerous substance. It may obtain access to the body either by inhalation or by absorption through the skin. Symptoms which have been noted in acute cases include headache, giddiness and tremor, accompanied sometimes by delirium and convulsions. There may also be muscular pains and weakness. As the lead is discharged in the exhaust gases, it would appear to be a potent source of lead poisoning in the motor industry and lead to pollution of the air in the streets. The United States Health Service appointed a committee to study the matter, and they reported[3] that drivers of cars using this fuel, in which the concentration of lead tetra-ethyl was not greater than 1 part to 1,300 parts by volume of petrol, showed no definite signs of lead absorption after exposures approximating to two years. In garages and stations in which the fuel was used, some absorption of lead was noticed, but the effect was slight. In places where lead tetra-ethyl petrol had been used as a motor fuel for two to three years, no definite cases were discovered of recognisable lead poisoning or other diseases resulting from the use of lead tetra-ethyl petrol. Two hundred and fifty-two adult males were examined. The committee found that stippling of the red cells gave results of value in the diagnosis of lead poisoning, and that the determination of lead in the fæces was also useful.

Toxicity and Fatal Dose. Lead poisoning occurs in two definite forms, *viz.*, the acute irritative and the chronic, and the cases below show that the toxicity of lead in the first form is very slight, but that in the chronic form lead is a very poisonous substance, dangerous both to life and to health. This arises from the fact that it is a very typical example of what is known as a cumulative poison, *i.e.*, once it has obtained access to the tissues it is removed from them with exceeding slowness.

[1] *Pharm. Jour.*, 1870, p. 426.
[2] Hunter, "Industrial Toxicology," 1944, p. 18.
[3] *Ind. Eng. Chem.*, 1926.

There are also the cases to be considered of subacute poisoning such as occur when lead is taken for abortifacient purposes (see under "Abortion").

Nothing certain is known concerning the *fatal dose* of acetate of lead. The facts already detailed show that it may be taken in comparatively large quantities without producing fatal effects. Thirty or forty grains have been given daily, in divided doses, without injury. Fiche[1] quotes a case of murder by means of white lead. The following cases, in some of which recovery took place in disadvantageous circumstances, prove that the acetate of lead is far from being a virulent poison.

Iliff met with an instance in which *an ounce* was swallowed in solution; the symptoms were pain in the abdomen resembling colic, with vomiting, muscular rigidity, and numbness. It was three hours before any remedies were used, and five hours before the stomach-pump was employed; but the person recovered. In the second case, also, an ounce was swallowed; sulphate of magnesium was freely exhibited, and the stomach-pump was used. On the following morning there was slight excoriation of the gums, which were white, with a sensation of heat in the throat; the bowels were relaxed, probably from the effect of the medicine. The day following there were pains in the legs and thighs, with restlessness and thirst. In a week the woman perfectly recovered. In a case which occurred to Alderson, a man swallowed an ounce of the acetate of lead in a drunken fit. There was violent vomiting, and the man recovered. A case of poisoning by carbonate of lead was reported by Snow in October, 1844, to the Westminster Medical Society. A child, aged five years, ate a portion not so large as a marble, ground up with oil. For three days he merely suffered from pain in the abdomen and costiveness. On the third night the child became rapidly worse, and there was vomiting. He died ninety hours after taking the poison, having passed some offensive motions of a greenish black colour (probably from admixture with sulphide of lead) before he died.

It is impossible to estimate the quantity of lead which has caused the fatal event in the chronic cases.

Blair Bell and his co-workers[2] have shown that there is great individual difference in tolerance to lead, and that the average amount of lead necessary to cause toxic symptoms varies from 0·1 to 0·3 gram. They also state that after intravenous injection lead is excreted almost entirely by the kidney, and that only minute amounts appear to be excreted by means of the bowel.

A daily intake of 1 to 2 mgm. of lead is generally accepted as likely to produce chronic lead poisoning, but this is considered too low an estimate by Kehve[3] and others, who found that the daily intake of slightly more than 2 mgm. per day, for a period of a year, failed to produce signs of poisoning.

Duration. Of the acute irritant cases there is little to be said. The symptoms are frequently delayed because the salts have but a feeble irritating power, and are easily influenced by the contents of the stomach. They rapidly subside on treatment, but occasionally in a few hours, or more commonly in a few days, these symptoms are followed by some of the symptoms of chronic lead poisoning.

Of lead which has been absorbed in the body, part is excreted (mainly in the urine) and part is deposited in the bones. The mechanism of deposition in the bones is probably the same as that for the deposition

[1] *Deut. Zeitsch. f. d. gerichtlich Mediz.*, 1926, 8, 735.

[2] *Lancet*, October 17th, 1925.

[3] Kehve and others, quoted by Jacobs, M.B., *Anal. Chem. of Lead*, [illegible] *Science Pub.*, New York (1941).

of calcium and is influenced by the same factors. Hence the relative amounts excreted and deposited vary very much. The symptoms of lead poisoning are due to the *circulating* lead. Hence there may be considerable amounts of lead harmlessly deposited in the bones, and yet no symptoms of lead poisoning may be evident ; if, however, conditions should favour withdrawal of lead from the bones (acidosis ; excessive activity of parathyroids, calcium and vitamin D deficiency, etc.), symptoms of lead poisoning may again become manifest. A case has been reported in which rapid mobilisation of lead from the bone marrow led to the death of a patient.[1]

In the chronic form it is impossible to estimate the duration either of the latent period—*i.e.*, the period during which minute doses are entering the body, but have not produced symptoms—or of the symptoms themselves when they have occurred. In some cases, under treatment and the avoidance of the poison, recovery may ensue in a few days from colic, or in a few weeks or months from wristdrop. The paralysis may be permanent, or death may intervene at any time from the effects on the brain, or from kidney complications.

This marked insidiousness of the symptoms often puts great difficulties in the way of discovering the cause, for though the symptoms and diagnosis may be unmistakable, the habits of the victim may afford no clue, owing to their regularity over long periods.

From a civil point of view it is impossible to put a limit to the possible action of lead, for it can start a condition of kidney trouble, which may persist and progress after lead has ceased to enter the system, and may ultimately cause death years later. See remarks on the Workmen's Compensation Act, Vol. I.

Symptoms. None of the salts of lead are very active poisons as irritants.

Christison states that he has given the acetate in divided doses to the amount of eighteen grains daily for eight to ten days without remarking any unpleasant symptom, except once or twice slight colic. When, however, the quantity taken has been from one to two ounces, the following symptoms have been observed : a burning, pricking sensation in the throat, with dryness and thirst ; vomiting and uneasiness at the pit of the stomach, sometimes followed by severe colic. The abdomen is tense, and the walls have been occasionally drawn in. The pain is relieved by pressure and has intermissions. There is generally constipation of the bowels. If any fæces are passed, they are commonly of a dark colour, indicative of the conversion of a portion of the lead into sulphide. The skin is cold, and there is a great prostration. When the case is protracted, the patient may suffer from cramp in the calves of the legs, pain in the inside of the thighs, numbness, and sometimes paralysis of the extremities. The affection of the nervous system is otherwise indicated by giddiness, torpor, and even coma. A well-marked blue line is noticed after a few days round the margin of the gums, where they join the teeth.

According to Blair Bell (*loc. cit.*), punctate basophilia of the red cells is the earliest and most constant sign of lead poisoning. There are also anæmia, nausea, vomiting, and colic. Damage to the kidney, with

[1] Brown, A., and Tompsett, S. L., *B.M.J.* (1945), 2 : 764.

diminution of the urine and albuminuria, is almost constant. Constipation is rarely seen in intravenous medication, nor is the blue line along the gums.

A woman, *æt.* 21, swallowed about three-quarters of a pint of Goulard's extract of lead (usually containing 24 per cent. of the subacetate), having begun with small doses. When first seen she was in great agony. There was severe colicky pain in the abdomen, and the muscles of the belly were drawn inwards. The pulse was feeble, there was trembling of the hands, and her body was in constant motion from severe suffering. There was heat in the throat and abdomen with intense thirst and a desire to vomit; but there was no actual vomiting or purging. A dose of sulphate of magnesium produced vomiting, and she recovered; but there remained obstinate constipation of the bowels.[1]

Of what may be called subacute symptoms, the persons who ate bread made with poisoned flour (p. 384) presented rather typical examples. In a few days they complained of a sense of constriction in the throat and the pit of the stomach, violent cramping pains round the navel, rigidity of the abdominal muscles, a dragging pain in the loins, and cramp with paralysis of the lower extremities. There was obstinate constipation, and the urine was scanty and of a deep red colour. Generally the pulse was slow and feeble; the countenance anxious and sunken, frequently of a peculiar livid hue; the tongue flabby; the gums marked by a deep blue line due to the formation of a sulphide of lead in the gum. The surface was cool, and there was a general arrest of the secretions. Sickness was not a uniform symptom, and even when it existed at first it speedily subsided. The mental faculties were undisturbed. Not one of the cases proved fatal, but among the more aggravated there was great prostration, with collapse, livid countenance, universal cramps, numbness, and other alarming symptoms. After apparent convalescence some of the symptoms returned in a more aggravated form without obvious cause, and for a long time the patients were out of health. Purgative medicines were found most effectual in the treatment. The quantity of acetate of lead taken by each person could not be determined, as, on analysis, the samples of bread were found to be unequally impregnated with the substance.

The symptoms of **chronic lead poisoning** are fully described in text-books of medicine and do not require detailed notice here. A few points must be considered, however.

The recognised symptom groups are as follows:—

1. Alimentary—Colic and constipation.
2. Referable to the peripheral nerves—Wristdrop.
3. Cerebral affections (lead encephalopathy).

In making a diagnosis of lead as the cause of any of these symptom groups, the blood should be examined for punctate basophilia of the red cells. The blue line on the gums is also looked for. In its absence, or in the absence of a clear history of exposure to lead, the cause of the symptoms is often overlooked. Of this lead line it must be noticed (*a*) that it consists in small bluish-coloured dots when examined with a lens; (*b*) that it occurs on the *edge of the gums*, not *on the teeth*; (*c*) that it only occurs on the gums of those teeth which have caries or dirt at the junction of gum and tooth; it is absent with good teeth kept cleansed;

[1] *Lancet*, 1860, 1, p. 33.

it must be looked for near to stumps or decayed teeth ; (*d*) it appears within a week in circumstances of dirt and decay favourable to its production, and (*e*) the shortest time in which it disappears is three weeks. A somewhat similar line may be seen in cases of mercury and copper, iron and silver poisoning [so it is alleged]. In unhealthy mouths utterly innocent of the tooth-brush a dark line often mistaken for a lead line by careless observers is sometimes seen, but a careful examination as to dotted appearance, colour, and position on the *gum* will exclude this.

The urine should be chemically examined for the presence of lead.

The colic requires to be differentiated from other sources of acute abdominal pain.

In addition to pain there are obstinate constipation, retraction of the abdominal parietes, loss of appetite, thirst, fœtid breath, and general emaciation, with paralysis of a peculiar kind affecting the extensor muscles, and causing a dropping of the wrist, or showing itself in a general paralysis of the limbs. The paralysis is peculiar, and at first affects almost exclusively the extensor muscles at the back of the forearm, so that the patient loses the power of raising the hand from the wrist, hence the name " wristdrop " applied to this form of paralysis. Squinting and amaurosis are occasional symptoms.

Cerebral symptoms are commonly observed and include intense headache, accompanied by fits closely resembling those of uræmia, and inasmuch as cirrhotic kidney is often caused by chronic lead poisoning, it is possible that these fits may be uræmic in some cases. Optic neuritis may be present ; if acute, it points more directly to lead ; if associated with much hæmorrhage and of a chronic type (albuminuric retinitis), it points more to cirrhotic kidney, which, however, may have been started by lead. In 1910 Dr. F. Smith met with a case of acute glosso-labio-paralysis occurring in a man after only three weeks' work among lead.

Works on medicine must be consulted for further details.

Treatment. In an *acute* case the stomach must be washed out, and if the patient is not already vomiting, a simple emetic must be given, also stimulants to combat shock and depression if present (though they are rarely seen). A brisk purge should then be administered, preferably of the sulphate and carbonate of magnesia. The sulphate and carbonate of lead are both insoluble salts, and may thus be expelled. Sodium thiosulphate has also been recommended as helping the formation of insoluble lead sulphide.

For the *chronic* cases removal of the source of the poison is the first essential. Potassium iodide has been used in the treatment of lead poisoning for over a century, and chemical analysis shows that it undoubtedly increases the excretion of lead. It withdraws lead for this purpose from that deposited in the bones, and may therefore precipitate an attack of lead poisoning long after exposure to lead has ceased.[1] This, incidentally, may be of service in the diagnosis of a doubtful case.

Aub and others[2] suggest that a diet low in calcium should be given, and advise the administration of acids and their ammonium salts, sodium bicarbonate and potassium iodide, and of parathormone. The treatment requires careful control (which is, however, easier than with potassium

[1] Oliver. Lead poisoning, London, 1914.
[2] *Medicine*, February-May 1925.

iodide) and these measures, designed to increase lead excretion, should never be used during colic or other toxic phases. In the presence of such symptoms, measures are taken to favour storage of lead—administration of a high calcium, high vitamin-D diet. In any case, periods of increased elimination at a rate which does not produce toxic symptoms are alternated with periods of storage. Eventually the readily mobilised lead is eliminated and the patient should then be left permanently on a diet which favours retention of lead in the bones.

Morphia and atropine or tincture of belladonna are useful for lead colic.

Post-mortem Appearances. Except for the gums and stomach, there is nothing characteristic without careful chemical analysis. In one case the mucous membrane of the stomach was much inflamed and of a dark red colour throughout. Lead could not be detected in the contents or tissues of the stomach, nor in the matter vomited. It is remarkable that in this case so small a quantity should have proved fatal without exciting any marked symptoms of irritation in the first instance.

In one acute case of poisoning by the acetate related by Kerchhoffs, the mucous membrane of the stomach was removed in several places, especially near the intestinal opening; and most of the intestines were in a highly inflamed state. In a trial for murder by this substance,[1] the stomach and intestines are stated to have been found inflamed, and there were dark spots on the former. In animals, according to Mitscherlich, when the dose is large, the mucous coat of the stomach is attacked and corroded; this change appears to be purely chemical, and takes place in all parts of the body with which the salt of lead comes in contact. If given in a small dose, it is decomposed by the gastric secretions, and exerts no corrosive power on the mucous membrane. When acetate of lead was given mixed with albumen and dissolved in acetic acid, death took place with great rapidity; but on inspection the stomach was found uncorroded. This corrosive action is a property of the neutral salt, and is not manifested when the dose is small, or when the poison is mixed with the acid.

Analysis. *Separation from gastric contents, tissues, snuff, etc.* The organic matter may conveniently be destroyed by the method described under "Arsenic" (p. 441), using minced tissue, or, in the case of fluids, after evaporation to a thin syrup. By this means the lead is removed from combination with proteins, etc. (in which form it responds badly to the usual tests, even when sufficiently concentrated) and is converted to chloride. Since lead chloride, though moderately soluble in hot water, is only slightly so in cold, it is essential to filter the final mixture while hot. The filtrate, which is usually strongly acid, should be almost neutralised with ammonia or sodium hydroxide, and treated with hydrogen sulphide. Any lead present will be precipitated as black lead sulphide which may be collected by filtration (or, better, by centrifuging). The lead sulphide should then be dissolved in a little warm dilute nitric acid, the solution evaporated to dryness, and the residual lead nitrate dissolved in water for further tests.

An alternative method consists in incinerating the dried tissue or evaporated fluid, dissolving the ash in nitric acid, evaporating the solution

[1] *R.* v. *Edwards*, C. C. C., November, 1843.

to dryness, redissolving the residue in water, and precipitating lead by means of hydrogen sulphide. The precipitate is then treated as above. Since the nitric acid may fail to extract the whole of the lead from the ash (*e.g.*, any present as sulphate) it is advisable to make a second extract of any insoluble material with an ammoniacal solution of ammonium acetate. This extract is treated with hydrogen sulphide as usual. The incineration method suffers from the disability that lead may be lost during the process since a number of its compounds are volatile. Moreover, the carbon present during the incineration may cause the production of metallic lead which may then alloy with the platinum and, in extreme cases, even fuse a hole in the dish. It is therefore advisable to accelerate the destruction of organic matter by the repeated addition of small quantities of concentrated nitric acid.

Detection and Estimation in Urine. (1) The urine, preferably about a litre in volume, is evaporated to dryness, and the residue is fused in a crucible with sodium nitrate until it is quite white. The mass is then extracted several times with *hot* dilute hydrochloric acid, the extract always being filtered hot. From the combined extracts, lead is precipitated by addition of hydrogen sulphide solution, and the precipitate is treated as already described.

(2) Fairhall [1] finds that lead is precipitated from urine almost completely by ammonia. The urine is treated with $\frac{1}{10}$ of its volume of concentrated ammonia and allowed to stand overnight. The precipitate is then filtered off, washed with a little distilled water, and extracted several times with hot 0·5 N. hydrochloric acid. From this solution the lead is, as before, precipitated by hydrogen sulphide.

(3) If lead is present in sufficient amount, it may be estimated after separation by one of the above methods, by precipitation as sulphate. The final solution of lead nitrate, obtained by solution of the lead sulphide in dilute nitric acid, is treated with an excess of dilute sulphuric acid. The precipitated lead sulphate, after standing for twelve to twenty-four hours, is filtered on a Gooch crucible, washed, dried, and weighed. One gram of precipitate corresponds to 0·6832 gram of metallic lead

Small amounts of lead may be estimated in the hydrochloric acid solutions obtained during the preliminary treatment by use of the lead electrode.[2] This method, for details of which the original paper must be consulted, is exceedingly delicate. Its use has shown the presence of traces of lead (about 0·05 mg. per litre) in the urine of normal persons.

Tompsett and Anderson[3] have developed an excellent colorimetric method for the determination of lead in tissues and excreta. The original paper should be consulted for details of the method which, in outline, is as follows. The material is dried and ignited in a silica dish (sodium pyrophosphate being added to soft tissues, but not to urine, fæces, or bone). Ignition is completed by heating with a little concentrated nitric acid. The ash is dissolved in hydrochloric acid, the solution is made slightly alkaline with sodium citrate and ammonia, and sodium diethyldithiocarbonate solution is added. The water-insoluble lead complex formed with this reagent is extracted with ether. The ether extracts

[1] Fairhall, *J. Biol. Chem.*, 1924, 60, 485.
[2] Millet, *Tr. Faraday Soc.*, 1929, 25, 147 ; *J. Biol. Chem.*, 1929, 83, 265.
[3] *Biochem. J.*, 1935, *29*, 1851.

are evaporated, organic matter in the residue is destroyed with sulphuric-perchloric acid mixture, the digest is treated with glacial acetic acid and ammonia and then made to a standard volume with water. An aliquot of this digest containing 0·01 to 0·02 mg. lead is treated with potassium cyanide, and diphenylthiocarbazone in carbon tetrachloride solution is added. The carbon tetrachloride layer, containing the lead complex (pink) is removed, excess reagent (green) is extracted with KCN solution, and the pink colour is compared with a standard. With suitable modification, the method is also applicable to blood.

In subjects with no known occupational exposure to lead Tompsett and Anderson reported a mean daily excretion of 0·05 mg. lead (range 0·045 to 0·07) in the urine and 0·22 mg. in the fæces ; the intake of lead from food and water was about 0·25 mg. per day. Tissues taken post-mortem from patients with no known occupational exposure to lead all contained traces of the metal ; liver, kidney and spleen averaged about 1·5 mg. lead per kilo, whilst bone averaged about 8 mg. per kilo (range 1·57 to 14·7).

Cholak and Bambach,[1] in over 1,000 persons with no occupational lead risk, found a mean of 0·027 mg. lead per litre of urine and 0·28 mg. per day in fæces. Figures ten times as large were found for 86 men engaged in making white lead.

Tests for Lead Salts in Solution. (1) With less even than 1 mg. of lead per 100 c.c. of solution, hydrogen sulphide or ammonium sulphide produces a black precipitate, soluble in *hot* dilute hydrochloric or nitric acid (with nitric acid sulphur is liberated, forming a pasty mass which may be dark coloured from undissolved lead sulphide). Hot, concentrated nitric acid converts it to the white, insoluble lead sulphate. (2) Potassium iodide added to a cold solution of a lead salt produces a yellow precipitate of lead iodide ; this dissolves in hot water, forming a colourless solution, from which, on cooling, it is deposited in the form of glistening golden crystals. Lead iodide is also soluble in sodium or potassium hydroxide solution. (3) Sulphuric acid, or sodium sulphate, gives a white precipitate of lead sulphate, insoluble in nitric acid, but soluble, on long boiling, in hydrochloric acid, or in excess of potassium or sodium hydroxide. (4) Sodium (or potassium) hydroxide gives a white precipitate easily soluble in excess of the alkali. (5) If a piece of zinc, wrapped with platinum wire, is dropped into the solution (best acidified with acetic acid), lead is at once deposited in crystalline form on the zinc. Alternatively the lead solution may be placed in a platinum dish or capsule, and a zinc rod dipped into it so as to touch the platinum. (6) Zinc alone will gradually precipitate lead as a dark spongy mass, even when the solution is contained in a glass vessel.

Tests for Special Salts. Lead Acetate. (1) Note the crystalline appearance and solubility (p. 384). It gives a clear solution with distilled water, but when it is dissolved in tap water the solution is usually cloudy, since the sulphates and carbonates present form insoluble lead salts. (2) Heated in air, it first melts and then resolidifies ; on further heating it changes in colour owing to the formation of yellow or orange-coloured litharge. (3) Heated in absence of air (*e.g.*, in a narrow tube) or with charcoal, it forms a black mass, consisting of metallic lead and charcoal.

[1] *J. Indust. Hyg.*, 1943, *25*, 47.

and a vapour is given off which smells of acetic acid and acetone. (4) The aqueous solution gives the general tests for lead salts. (5) The substance gives the general tests for acetates (p. 603).

White Lead. (1) A white powder, amorphous, insoluble in water. (2) It is dissolved by dilute nitric acid or, to a smaller extent, by hot dilute hydrochloric acid, in each case with effervescence—the gas evolved is carbon dioxide, and turns lime-water milky. (3) When heated it leaves a residue of litharge. (4) Its solution in acid gives the general tests for lead salts.

Poisoning by Arsenic

Source and Method of Occurrence. More interest attaches to arsenical poisoning, perhaps, than to any other form of poisoning, owing to the fact that it is the one most commonly chosen by murderers.

Compounds of arsenic are in common use in arts and manufactures, and are consequently prepared in large quantities. The following arsenical compounds have been known to give rise to symptoms of poisoning, but there is no substance which contains arsenic which is not poisonous :—

1. Arsenious oxide or arsenious acid (probably the most common form), also known as white arsenic, or, more commonly but erroneously, as arsenic.
2. Arsenites of potassium and sodium.
3. Copper compounds of arsenic or arsenical pigments.
4. Arsenic acid and arsenates.
5. Sulphides of arsenic Orpiment (yellow) and Realgar (red).
6. Chlorides of arsenic.
7. Arseniuretted hydrogen (arsine).
8. Other Arsenical Gases and Toxic Smokes.
9. Organic compounds of arsenic—cacodylates, atoxyl, soamin, salvarsan (606), etc.

1. **White Arsenic, Arsenious Acid.** The term *white arsenic* is commonly applied to the arsenious oxide or acid. It is seen as a white powder, visibly crystalline in a strong light, or when viewed with a lens. It is also met with in opaque, brittle white masses, resembling enamel. It is called an acid from its power of combining with alkalies, but it possesses only a feeble acid reaction when dissolved in water. It is often described as having an acrid taste, but this does not appear to be correct ; a small quantity of it has certainly no appreciable taste, a fact which may be established by direct experiment. It would appear, from numerous cases on record, that it has been unconsciously taken in fatal quantities, in all descriptions of food, without exciting any perceptible sensation on the tongue.

In 1880 a baker's boy of Saint Denis, wishing to avenge himself on his employer, mixed arsenic with the bread and poisoned 270 people.[1] In 1867 at Wurzburg 373 persons were poisoned in a similar manner (cited by Vibert).

At Bradford, in 1858, seventy persons suffered, and seventeen died, from the effects of eating peppermint lozenges, adulterated with white arsenic, in mistake for "daft" (sulphate of lime), which was at one time commonly used for the adulteration of cheap lozenges.

[1] Brouardel, "Les Intoxications."

In September 1930, an outbreak of arsenic poisoning occurred amongst children in Congleton, and on inquiry it was found that in a factory at Stoke-on-Trent quantities of sweets had been dusted with a white powder supposed to be rice starch but which was really composed of white arsenic. Samples of the sweets which were analysed were found to contain as much as two-thirds of a grain of arsenic in each sweet. Fortunately, although there were many cases of acute illness, there was no loss of life, which, in view of the heavy contamination, is rather extraordinary.

Most of those persons who have been criminally or accidentally killed by arsenic have not been aware of any taste in taking the poisoned substance. In cases in which the powder has been taken in *large* quantity, it is described as having a *roughish* taste.

Mixtures of white arsenic, soft soap, and either sulphur or tar-water, are largely used in agricultural districts for dipping sheep. These have caused death, with the usual symptoms of arsenical poisoning, in several instances.

In 1874, the Coombs family, consisting of eight persons—the father, mother, and six children—were all poisoned, four of them fatally, by drinking water from a bucket which had contained an arsenical sheep-dipping composition.

In 1878, some remarkable cases of poisoning by the external use of violet-powder mixed with white arsenic occurred at Loughton, in Essex, and in other places. Though a number of children died suddenly with somewhat similar symptoms, in two cases only were *post-mortem* examinations and analyses of the viscera conducted. One child, a female, was at birth described by the nurse as a healthy child. It was washed, as usual, shortly after birth, and was then dusted all over with the violet-powder, a sample of which was subsequently ascertained to contain 38½ per cent. of white arsenic. Later on in the day the powder was again applied to the private parts of the body. On the second day powder was again applied four times. On this day there was very marked redness and inflammation of the skin, and this led the nurse to apply the powder unusually freely, especially over the stomach, where the redness was most noticeable. On the third day the skin was intensely red, and appeared in parts—especially about the navel, the vagina, and the anus—of a yellow and unhealthy hue. In consequence of it being suspected that the violet-powder contained some irritant substance, its use was discontinued, and common starch-powder was used instead. On the succeeding day the eruption became very much worse, and in some parts of the skin commenced sloughing. On the tenth day of the child's life, and on the sixth day from the last application of the violet-powder, the child died from exhaustion. A *post-mortem* examination, made five days after death, revealed nothing marked in the condition of the viscera. From the exhumed body six and a half grains of arsenic were obtained by Tidy.

Arsenic has also caused death by being inhaled in the form of vapour.

In a case which was the subject of a trial at the York Lent Assizes, 1864, the accused placed some burning pyrites, containing arsenic, at the entrance of the door of a small room in which there were eight children, including an infant in a cradle. The accused was found guilty of manslaughter.

2. **Arsenites of Potash and Soda, Liquor Arsenicalis (Fowler's Solution).** There is one case recorded in which Fowler's solution has destroyed life. A woman took half an ounce (= two grains of white arsenic) in divided doses during a period of five days, and died from the effects. There was no vomiting or purging, but after death the stomach and intestines were found inflamed.

Arsenite of sodium is as poisonous as arsenite of potassium.

In December 1857, 340 children belonging to an industrial school near London were poisoned by this compound. It had been incautiously used for cleansing a steam boiler, and had thus become mixed with the hot water which was drawn

for the breakfasts of the children. The dose of arsenic taken by each child was about one grain. All recovered, although some suffered severely. In the winter of 1863, a man died under symptoms of acute poisoning by arsenic owing to his having drunk beer out of a pot which had contained this *patent* cleansing liquid.

Some "weed-killers" are solutions of arsenite of sodium. They may contain from 14 to 40 per cent. of arsenious oxide. Weed-killers are often sold in powder form. In the Greenwood case, the "Eureka" weed-killer which was used contained 60 per cent. of arsenious acid coloured with phenolphthalein.

In 1891, a man named Wesley died, and four members of his family were made seriously ill, by drinking gooseberry wine stored in a cask in which a weed-killer had been sent out. Each fluid ounce of the wine was found by Sir T. Stevenson to contain nearly six grains of arsenious acid. Several other deaths occurred about the same time from these weed-killers; and in 1891 an extensive series of cases of poisoning by sugar into which a weed-killer had leaked during transit was recorded by Thom.[1] In April, 1920, an outbreak of poisoning occurred from the contamination of sugar by arsenical weed-killer during transit. Forty-four persons were affected.

Wood preservative solutions sold for killing insects infesting wood are usually solutions of white arsenic, and as they are uncoloured, they are a source of danger.

Fly-water is a name applied to solutions of various arsenical compounds in water. Mixtures of this kind are formed by dissolving one part of arsenite of sodium and two parts of sugar in twenty parts of water. Paper soaked in this solution, and dried, is used for poisoning flies; and perhaps this is the safest form in which arsenic can be used for such a purpose.

According to Sir T. Stevenson's analysis, an ordinary fly-paper of about the area of a page of this book contains five grains of arsenic. Wilcox has obtained very nearly the same quantities. Fly-papers formed a very prominent feature in the Maybrick and Seddon cases. Fly-powder is a dark-coloured mixture of metallic arsenic and arsenious acid.

3. **Copper Arsenite, Scheele's Green, Emerald Green, etc.** This is the only other arsenite which is met with in commerce and the arts, and it constitutes, wholly or in part, a great variety of green pigments, known as emerald green (aceto-arsenite of copper) employed for paper-hangings, Mineral green, Brunswick, Schweinfurt, or Vienna green. It is thus found in the form of oil-paint, in cakes of water-colours, wafers, adhesive envelopes, or, rarely, spread over confectionery, and lastly, and most abundantly, in various kinds of green decorative papers used for covering the walls of rooms.

It was used to colour blanc-mange for a public dinner. It led to death under the usual symptoms, and the persons responsible were convicted of manslaughter and sentenced to imprisonment.[2]

Taylor found it on some loaves of bread which had been placed on shelves painted with arsenical paint. Painters using arsenical colours have also suffered.

Wall-papers covered with the loosely adhering aceto-arsenite of copper were formerly, from their cheapness as well as their brightness of colour, extensively used in dwellings. This pigment contains 59 per cent. of arsenic, and from some

[1] *Lancet*, 1891, 1, p. 900.
[2] *R.* v. *Franklin and Randall*, Northampton Sum. Ass., 1848.

of these papers in the unglazed state the noxious material may be easily scraped or removed by friction. A square foot may yield from twenty-eight to seventy grains of the arsenical compound, and in rooms exposing five or six hundred square feet arsenic is thus liable to be distributed in the state of a fine dust or powder through the air of a room. This poisonous dust has been detected on books, picture-frames, furniture, and cornices of rooms thus papered.

Similar symptoms of poisoning have arisen from a person occupying a room filled with stuffed birds and animals in the preservation of which an arsenical compound had been used. Arsenic was found in the dust of the room and on the furniture.[1]

The arsenical compound has also been much used for colouring artificial flowers, wreaths, and tarlatan dresses. Dressmakers occasionally suffer seriously from this form of poisoning.

Two women were employed to make some green tarlatan into ball-dresses. They noticed an unpleasant smell and taste, and their eyes were affected during the performance of the work. The symptoms from which they suffered were swelling of the eyelids, congestion of the conjunctivæ, and copious secretion of tears. The one most affected experienced on the second day salivation, with an unpleasant taste in the mouth, cramps in the limbs, great thirst, restlessness, and difficulty of breathing. These symptoms lasted in one patient eight, and in the other fourteen days.

Riedel, who describes these cases, suffered severely from a similar train of symptoms for several days, as a result of handling the poisoned dresses for the purpose of analysis. He found that the stuff contained 13 per cent. of its weight of arsenic.[2] To this list may be added the case of a lady who suffered severely from symptoms of arsenical poisoning by reason of her having worn, on one occasion only, a dress of this description. Paper used for adhesive envelopes, for wrapping confectionery, children's food, isinglass, chocolate, etc., is also occasionally coloured with arsenical pigments.

These pigments are now rarely used, and cases of poisoning from such sources are almost matters of history, but they require to be kept on record as possibly explaining some mysterious cases of illness.

4. **Arsenic Acid and Arsenates of Sodium and Potassium.** Arsenic acid was formerly employed in the manufacture of magenta, rosaniline, and other colours from aniline. There is reason to believe that the colour is sometimes sent into the market contaminated with arsenic. Rieckher found from 1 to 7 per cent. of arsenic acid in the red colours supplied by good manufacturers, and frequently arsenious acid was also present.[3] As these red compounds are used for giving a beautiful red colour to liqueurs, syrups, raspberry vinegar, and sugar sweetmeats, there is a possibility that accidents may occur from their use. They are rich in tint and very inexpensive.[4] The injurious effect of several of these coal-tar pigments when used for dyeing socks and gloves has been ascribed to the arsenic contaminating the dyes, but perhaps in most instances incorrectly. Several varieties of printers' ink have been shown to contain arsenic.[5] But few cases of fatal poisoning by the *arsenates* are recorded.

5. **Sulphides of Arsenic : Orpiment and Realgar.** Orpiment, or yellow arsenic, owes its poisonous properties to the presence of a variable proportion of arsenious acid. Orpiment is much employed in the arts, in painting, dyeing, paper staining, and even in the colouring of toys, but is not often used as a poison. In December 1859 six persons suffered from the usual symptoms of poisoning by arsenic owing to their having

[1] "Ann. d'Hyg.," 1870, 1, 314.
[2] Husemann, "Jahresber. der Toxicol.," 1871, p. 525; 1872, p. 480.
[3] *Med. Times and Gaz.*, 1870, 1, 617.
[4] *Ibid.*, 1870, 1, pp. 46, 84.
[5] *Analyst*, April 1927.

eaten *Bath buns*. It was found that a confectioner at Clifton had used, as he supposed, chrome yellow (chromate of lead) to give the buns a rich yellow colour, but the chemist to whom he applied had ignorantly supplied him with orpiment. Orpiment mixed with lime may be used in the processes of fellmongering and tanning for the purpose of removing the wool of the sheep from the hide. Sir T. Stevenson saw severe injuries result from the mixture coming accidentally into contact with the skin of those engaged in the trade.

In one case, two persons partook of some porridge in which orpiment had been put by mistake for turmeric. One, an old man, died in twenty-two hours; the other, a boy, recovered. On inspection there was violent inflammation of the gullet and stomach, the mucous coat of the latter being softened and thickened. There was a gangrenous spot, one inch in diameter, in the gullet, and another in the stomach, three inches in extent.[1]

Orpiment is much used in India and other parts of the East both as a medicine and as a poison. Orpiment and realgar (another sulphide of arsenic) are sold openly in India, and are used as depilatories. Orpiment has been known to cause death by *external* application as a depilatory.

In *R.* v. *Stuart*[2] a novel question arose respecting this compound. There was some reason to believe that the deceased woman had died from the effects of arsenic administered in confectionery. White arsenic was found in the stomach, and a question was put by the judge, as well as by the counsel for the accused, whether the confectioner might not have used yellow arsenic by mistake in order to give a colour, and this yellow arsenic have been converted in the deceased's body in twenty-four hours into white. It need hardly be remarked that the yellow colour is an essential character of orpiment. White may be converted into yellow arsenic in the dead body, but yellow cannot be thus changed into white arsenic.

6. **Chloride of Arsenic.** A solution of arsenic in dilute hydrochloric acid, containing 1 per cent. of arsenious acid, was formerly an official preparation. It is highly poisonous. A woman took three doses in a period of twenty-four hours. The quantity of arsenic thus taken was not more than the *tenth part* of a *grain*, and yet the symptoms which followed were of a severe kind, resembling those of chronic poisoning. The medicine was withdrawn, and the patient slowly recovered. It seems that she had not taken arsenic previously; and there was obviously a peculiar susceptibility to the effects of arsenic. The quantity taken was very small to produce such alarming effects. The dose is from three to ten minims.

7. **Arseniuretted Hydrogen (Arsine) (AsH_3).** This is a gaseous compound of arsenic formed by the action of nascent hydrogen on reducible arsenical compounds, and is therefore liable to be evolved in any industrial process involving the production of hydrogen, for arsenic is a contaminant in many industrial substances in which its presence may not be expected. The gas is colourless but has a garlic odour. It is dangerous when inhaled and may cause death in a concentration of 300 parts per million of air. It produces hæmolysis of the red cells of the blood and is probably the

[1] Wharton and Stille, "Med. Jur.," p. 434.
[2] Lewes Lent. Ass., 1863.

main hæmolytic poison encountered in industry.[1] Early symptoms include headache and nausea and passing of blood in the urine. Jaundice develops later associated with symptoms of arsenic poisoning. Multiple neuritis has also been produced.[2]

On post-mortem examination hæmorrhage into the mucous membranes, fatty degeneration of the heart, liver and kidneys, a yellow discoloration of the organs, inflammation and œdema of the lungs may be found. Laking of the blood and signs of secondary anæmia may be observed.

In 1892 two deaths occurred at Llanelly from the inhalation of an impure form of this gas. Two men dissolved in hydrochloric acid, in an open tube, a mixture of metals containing 0.8 per cent. of arsenic and some antimony. The gases liberated would contain both arseniuretted and antimoniuretted hydrogen. One of the three men engaged in the operation was exposed to the fumes for ten or fifteen minutes. About six hours after he was seized with diarrhœa and severe abdominal pains; on the following day he was jaundiced; on the sixth day he seemed better; and on the seventh day paralysis of the arms and legs supervened, and he died suddenly. One of the assistants suffered from similar symptoms, and died on the tenth day.[3]

It has been suggested that it is by means of this gas that some of the fabric and wall-paper cases may occur. Thus two theories hold the field :—

(1) That of Forster, who believes that the poisoning is due to detachment of solid particles from the stuffs which float in the air and enter the body by means of the respiratory passages; and (2) that proposed by Selmi, who considers that toxic gases may be formed from the pigments by the action of microbes. Some experiments undertaken by Gosio[4] lend a good deal of support to this latter view. This observer made nutrient media of potato paste, impregnating them in some cases with arsenious acid, and in others with various arsenical pigments: the nutrient media thus prepared were exposed for some days to a constant current of air, which was drawn over them by means of an aspirator. The air, after it had passed over the paste, was made to bubble through a solution of nitrate of silver, which would detain all volatile arsenical compounds which might have been formed. As a result of this first rough experiment the author found that many moulds and some fission fungi developed on the arsenical media, and that very appreciable quantites of volatile arsenic compounds were given off. Proceeding then to isolate the different species which developed, and testing the action of each on fresh sterile prepared media, he found that only two species—*Mucor mucedo* and in a less degree *Penicillium glaucum*—were able to split up the solid arsenical compounds as above described. From a great number of experiments he came to the following conclusions :—(1) *Mucor mucedo* will grow well in presence of a notable quantity of arsenic; it seems to grow more luxuriantly in these than in normal conditions. (2) Many fixed compounds of arsenic, and among them the green pigments ordinarily used, are changed by the growth of these moulds into gaseous bodies, among which is certainly arseniuretted hydrogen. (3) The sulphides are not decomposed like the oxygenated compounds, but their presence in the culture media seems in no way detrimental to the growth of the moulds. (4) In certain conditions of light, moisture, and temperature, it is possible to obtain arsenical gas by the growth of *Mucor mucedo* (and perhaps other mucorinæ) on tapestry coloured by either Scheele's or Schweinfurt's green. Thus, without denying the possibility of occasional poisoning by detached particles, Gosio considers he has made out a strong case in favour of the gaseous theory of arsenical poisoning.

These fungi may be utilised for toxicological purposes in the detection of arsenic.[5]

[1] Guelman, *J. Ind. Hyg.*, 7 : 6, 1925.
[2] Muehlberger, C. W., *et alia, Journ. Ind. Hyg.*, 10 : 137 (1928).
[3] *Public Health*, 1892, vol. 4, p. 317.
[4] "Laboratory Reports of the Public Health Department," Rome, September 1891.
[5] *Lancet*, 1903, 2, p. 1728.

8. **Other Arsenical Gases and Toxic Smokes.** A number of toxic arsenical gases such as the dichloroarsines, Lewisite, etc., have been described in connection with gas warfare. Full details of these may be obtained from the War Office publications on gas warfare.

9. **Organic Arsenical Compounds.**[1] The organic compounds of arsenic are much less toxic than inorganic arsenic compounds containing an equivalent amount of the element arsenic. An enormous number has been synthesised in attempts to find the ideal substances for combating various parasitic diseases, but relatively few have fulfilled the conditions necessary for therapeutic use. A satisfactory drug must, of course, be toxic to the parasites it is desired to attack (*in vivo*, if not *in vitro*), and it must be less toxic to the host. In other words, the ratio of the minimum lethal dose (from the point of view of the host) to the effective therapeutic dose must be as large as possible in order to allow the maximum margin of safety. The drug should, moreover, give rise to the minimum of undesirable side-effects, and should be as stable as possible lest, when kept, it decomposes to more toxic substances.

In spite of the vast strides made in recent years, it can still, unfortunately, be said that the perfect organic arsenical drug has not yet been obtained, and that none of those used to-day is entirely free from danger.

The organic compounds of arsenic, whether they contain trivalent or pentavalent arsenic, may be classified according to the type of organic radical with which the arsenic is combined.

(1) Aliphatic Series

The most important members of this group are the *cacodylates*.

Cacodylic Acid. $As(CH_3)_2.O.OH$, dimethyl arsenic acid, is a white crystalline substance, soluble in water, odourless, and relatively non-toxic. In 1862 it was used in chronic skin diseases and in tuberculosis, and much more recently it has been used, chiefly in the form of one of its salts, in the treatment of syphilis, disseminated sclerosis, malaria, chronic broncho-spirochætosis, etc.

The usual derivative used therapeutically is the sodium salt which contains 35 per cent. of metallic arsenic. It is deliquescent, and liable to contain small amounts of inorganic arsenic. It may be decomposed, to some extent, by the digestive juices when administered orally or rectally (arsenic being liberated) and so give rise to alarming symptoms—garlic taste, nausea, gastric pain, thirst, œdema of the eyelids, renal congestion with albuminuria. It is usually administered hypodermically in a dose of $\frac{1}{2}$ to 1 grain, but is recommended also in doses of 2 to 3 grains intramuscularly for seven to ten days, or in doses of 10 to 30 grains intravenously at four-day intervals. Its therapeutic effect depends upon the continuous action of the arsenic slowly liberated from the cacodylic acid. The greater part appears unaltered in the urine but part appears as inorganic arsenic. Overdosage with cacodylates causes the same toxic symptoms as arsenious oxide.

Cacodyl $(CH_3)_2As—As(CH_3)_2$, is a volatile colourless liquid, with a strong odour of garlic. It is very poisonous.

[1] Full information about the various organic arsenicals may be obtained from the Reports of the Salvarsan Committee, Special Reports Series, Nos. 44, 55 and 66, H.M. Stationery Office.

Disodium Methyl Arsenate, $CH_3AsO.(ONa)_2$, New Cacodyle, Neoarsykodile, Arsynal, **Arrhenal,** contains one methyl group less than the cacodylates, but, like them, contains pentavalent arsenic. It is soluble in about its own weight of water, but only slightly soluble in alcohol. In crystalline form ($+ 5H_2O$) it contains 27·35 per cent. of arsenic. It is used for the same purposes as sodium cacodylate, in doses of $\frac{2}{5}$ to 3 grains, orally or hypodermically. A dose of 1 grain was followed by vomiting and diarrhœa but no abdominal pain.

Tests. The cacodylates are not reduced to arsine by nascent hydrogen and therefore yield no metallic mirror when subjected to the Marsh test. Under certain circumstances[1] they give an orange-yellow deposit. They give no precipitate of arsenious sulphide when treated with hydrogen sulphide. They are, however, decomposed by long-continued boiling (six hours or longer) with sulphuric acid, and then give the ordinary arsenic reactions. They are not decomposed by the Fresenius method of destroying the organic matter of tissues (potassium chlorate—see p. 441)

Arrhenal is less stable than the cacodylates, and its solution, acidified and saturated with hydrogen sulphide, yields a yellow precipitate of arsenious sulphide.

Like most organic arsenicals, these compounds are extracted from the tissues by the method of **Vitali.**[2] The finely minced tissue is mixed with water, acidified with tartaric or hydrochloric acid, and evaporated to dryness. The residue is extracted three times with 90 per cent. alcohol, and the filtered extracts are distilled until all the alcohol has been removed. The aqueous residue, containing the organic arsenic compound, is then boiled with concentrated sulphuric acid, potassium sulphate, and a little potassium permanganate. The liquid is diluted when oxidation is complete, filtered if necessary, and saturated with hydrogen sulphide. The arsenic is precipitated as sulphide. Alternatively, the alcoholic extract, after removal of the alcohol, may be used for estimation of arsenic by Robertson's[3] or Lehmann's method.[4]

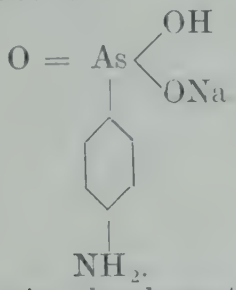
Lehmann's method (modified) is as follows: A suitable amount of material, in a 200 c.c. flask provided with a glass stopper, is mixed with 1 gram of powdered potassium permanganate and 10 c.c. of dilute sulphuric acid. After standing for ten minutes, with frequent rotation of the flask, the mixture is treated with five successive lots of 2 c.c. of concentrated sulphuric acid, the flask being rotated after each addition. When the reaction is over, the brown precipitate is dissolved by addition of hydrogen peroxide (5 to 7 c.c.), care being taken to avoid excess. The unavoidable slight excess of hydrogen peroxide is removed by diluting the mixture with 25 c.c. of distilled water and boiling for ten minutes. If necessary (to avoid reappearance of the brown precipitate owing to evaporation of the solution) the last trace of hydrogen peroxide may be removed by a drop of permanganate solution, the excess of which is in turn destroyed by a drop of oxalic acid solution. The colourless solution is then diluted with 50 c.c. of distilled water, 2·5 grams of potassium iodide are added, and the stoppered flask is allowed to stand in a cool

[1] Ganassini, *Boll. chim. farm.*, 1919, 58, 385.
[2] *Boll. chim. farm.*, 1901, 40, 657; 1903, 42, 641.
[3] *J. Amer. Chem. Soc.*, 1921, 43, 182.
[4] *Apoth. Zeitung*, 1912, 27, 545; *U.S. Pub. Health Rep.*; 1918, June 21st, 1003.

place for an hour. The liberated iodine is then titrated with standard sodium thiosulphate solution without the use of starch indicator ; 1·0 c.c. of $\frac{N}{10}$ thiosulphate is equivalent to 0·003748 grams of arsenic.

(2) Aromatic Series. A. Pentavalent Arsenic

Atoxyl (Soamin, Arsamin, Sodium arsanilate). In 1905 Thomas and Breinl, at Liverpool, showed that atoxyl (discovered by Béchamp in 1863 and wrongly believed to be arsenic anilide) possessed marked trypanocidal properties. The substance was then used by Koch in the treatment of sleeping sickness, but was found to be so toxic, producing injury to vision, optic atrophy, and even death, that its use was abandoned. The true constitution of atoxyl was shown by Bertheim to be that represented by the formula :—

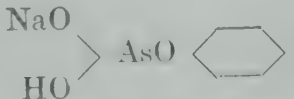


Sodium *p*–amino–phenylarsenate Atoxyl.

Apparently atoxyl is still used therapeutically to some extent, for though it has been found too toxic for use in syphilis it is an efficient trypanocide in *African sleeping sickness*. It is a white crystalline substance ($+ 4H_2O$), odourless, soluble in water, and containing about 25 per cent. of arsenic. Though sometimes given by the mouth, it is liable to be decomposed by the gastric juice with the liberation of inorganic arsenic. It is given hypodermically in doses of $\frac{1}{3}$ grains on alternate days, and intramuscular doses of 10 grains have been given at intervals of several days to a total of 100 grains.

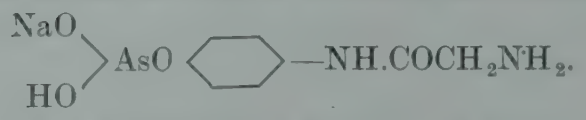
The toxicity of atoxyl is said to be about $\frac{1}{40}$ of that of arsenious oxide. Overdosage leads to blindness from atrophy of the optic nerve. Schlecht[1] reported a fatality from four hypodermic injections, within eight days, of a total of 36 grains of atoxyl. Death occurred on the second day after the last injection with lung œdema and paralytic phenomena. The *post-mortem* examination showed degenerative changes in the heart and liver similar to those in poisoning by arsenious oxide.

Atoxyl is largely excreted unchanged in the urine, but some is found in the fæces and sweat. The organs of a dog poisoned by atoxyl contained inorganic arsenic.

Acetyl-Atoxyl, Arsacetin,  $NH(CH_3CO)$, is white, crystalline, water soluble, and less toxic than atoxyl, which it resembles therapeutically.

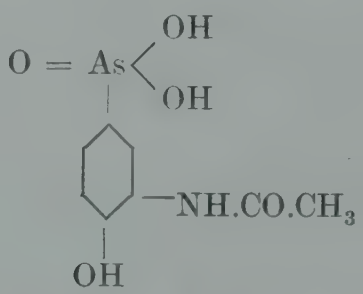
Tests. Atoxyl and its acetyl derivative respond to the Marsh and Gutzeit tests, and slowly to the Reinsch test. A solution, slightly acidified with hydrochloric acid and saturated with hydrogen sulphide, gives little or no precipitate in the cold, but on warming, yellow arsenious sulphide is precipitated. Atoxyl gives the various tests for primary aromatic amines.

Tryparsamide, Moranyl, sodium *n*-phenyl-glycinamide-*p*-arsenate.



Tryparsamide is a condensation product of atoxyl and glycine, and is largely used in the treatment of neurosyphilis and trypanosomiasis (sleeping sickness). It has a relatively feeble action on spirochætes, but is claimed to have exceptional powers of tissue penetration. Like other compounds containing pentavalent arsenic, it is probably decomposed slowly in the tissues with formation of trivalent arsenic. It does not produce the atrophy of the optic nerve which was noticed in the case of atoxyl. The usual dose is 1 to 4 grams weekly by injection, but as much as 7 grams has been given intravenously without ill effects.

Stovarsol, Kharophen, Fourneau 190, acetylamino-oxy-phenyl arsenic acid.



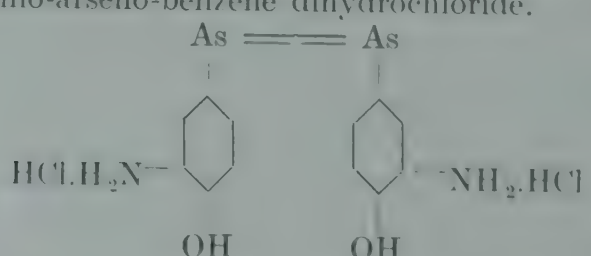
Stovarsol, introduced in 1922 by Fourneau and Levaditi, forms colourless crystals, almost insoluble in water and alcohol. It contains about 27 per cent. arsenic (as supplied commercially). It is readily absorbed from the alimentary canal, and is usually administered by mouth in doses of 4 to 8 grains (even more in treatment of yaws) per day for a few days. Its chief use is in the treatment of amœbic dysentery, but in France it is also used in the prophylactic treatment of suspected early syphilis. Although its toxicity is low, toxic symptoms are sometimes produced, and its continued use is said to lead to exfoliative dermatitis.

Acetyl Arsan, a diethylamine derivative of stovarsol.

Acetyl arsan, a crystalline substance soluble in water, is also used in the treatment of amœbiasis, and in the treatment of infantile syphilis. It is given intramuscularly or subcutaneously. The commercial solution contains 23·6 per cent. of the substance, equivalent to 0·55 grams of arsenic per cubic centimetre. (Dose, up to 5 c.c., weekly.)

B. Trivalent Arsenic

Salvarsan, Kharsivan, Arsphenamine, Arsenobenzol, Ehrlich's 606. Dihydroxy-diamino-arseno-benzene dihydrochloride.



[1] *Münch. med. Woch.*, 1909, 56, 972.

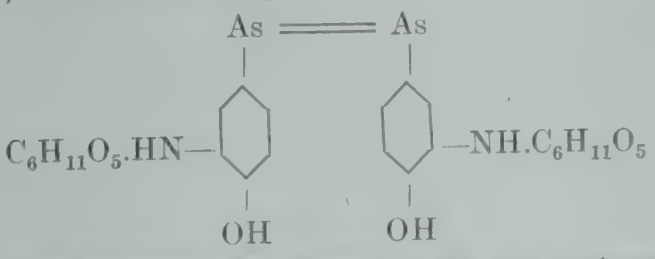
Salvarsan was the culmination of a long series of researches by Ehrlich and his collaborators, with the object of finding a *therapia magna sterilans* —a complete sterilisation of the tissues by a single injection of parasiticidal substance. This ideal has been attained only in relapsing fever, which is usually cured by a single dose of neosalvarsan, but in salvarsan Ehrlich produced a drug which, in spite of its many drawbacks still remains a potent arsenical agent for the treatment of syphilis.

Salvarsan is a pale yellow powder containing 32 per cent. of metallic arsenic. It is soluble in water (1 gram dissolves in about 5 c.c. of water), but the solution is acid and so irritant that it cannot be injected intravenously. Addition of sodium hydroxide (2 mols.) to the solution precipitates the free base which is insoluble in water. The suspension of this base can be used for intramuscular, but not intravenous injection. Further addition of sodium hydroxide (2 mols.) forms a disodium salt which is freely soluble, and can be given intravenously in dilute solution. This is the form in which salvarsan is usually administered, but since it is irritant, care must be taken to prevent its escape into the tissues. Excess of alkali must be avoided, or the vessels may be thrombosed. Both the free base and the disodium salt oxidise readily in air, with formation of toxic products ; the dihydrochloride is relatively stable, but oxidises slowly, and is therefore marketed in sealed tubes filled with nitrogen or other inert gas (no tube should be used without being first tested to prove the absence of leaks). The solution for injection is to be prepared immediately before use, with scrupulous attention to all details of the directions.

The intravenous injection of 0·2 g. or more of unalkalinised salvarsan nearly always causes death. (Such a catastrophe may result from mistaking salvarsan for neosalvarsan which requires no alkalinisation.)

Toxicity. For mice, weighing 20 gram, the maximum amount tolerated without a fatal result is about 0·125 gram per kilo. ; for rats the maximum non-lethal dose is about 0·08 gram per kilo. In man, the therapeutic dose varies from 0·1 to 0·6 gram at intervals of a few days. There seems to be a considerable variation in susceptibility to toxic effects, both in man and animals, even when technical errors in administration can be eliminated. It has, moreover, to be remembered that the drug, when used for therapeutic purposes, is not being administered to healthy individuals. According to the Salvarsan Committee of the Medical Research Council (1922, Special Report No. 66), salvarsan causes one death in 13,000 cases, excluding fatalities due to errors in administration.

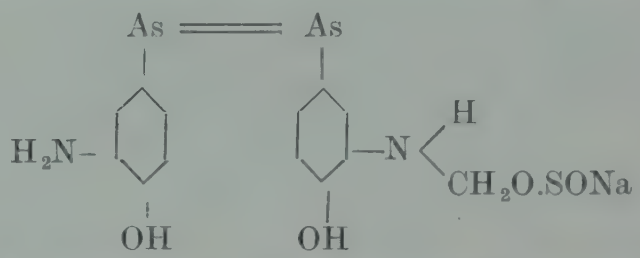
Stabilarsan, Arsenobenzol diglucoside.



Stabilarsan, a compound of salvarsan with two molecules of glucose, is freely soluble in water, and is claimed to be stable on exposure to air —and therefore less toxic than salvarsan.

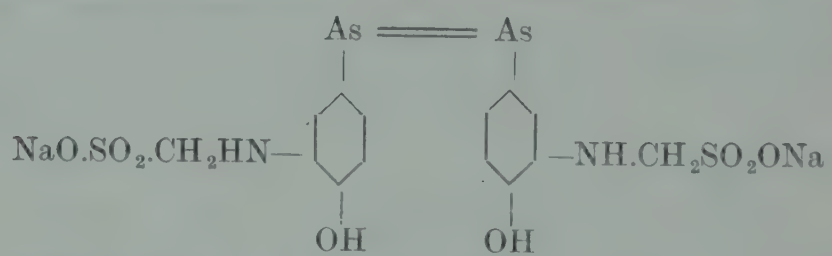
Silver Salvarsan is a compound of silver and arsenobenzol containing 22·5 per cent. of arsenic and 14 per cent. of silver. It is a brownish-black powder, easily dissolving in water to a solution suitable for intravenous injection. Its therapeutic and toxic effects are about twice as great as those of salvarsan.

Neosalvarsan, Neokharsivan, Neoarsphenamine, Novoarsenobenzol, sodium dihydroxy-diamino-arsenobenzene-methylene sulphonate. Ehrlich's 914.



Neosalvarsan is a yellow powder (rather deeper in colour than salvarsan), readily soluble in water to a solution neutral to litmus. In practice the amount of arsenic present varies somewhat, and must, according to the regulations, lie between the limits of 18 per cent. and 21 per cent. It is less active therapeutically than salvarsan, the dose being about 1·5 times that of salvarsan. Its toxicity, however, is more than correspondingly less—it has been reported as causing one death in only 130,000 cases, though in doses over 0·6 gram it causes one death in 5,000 cases. Neosalvarsan can be given either intravenously or intramuscularly; like salvarsan, however, it changes on exposure to air, so that it is distributed in sealed ampoules and its solution must be used as soon as prepared.

Sulpharsenol, Kharsulphan, Sulpharsphenamine, Sulpharsenobenzene, Dihydroxy-diamino-arsenobenzene dimethylene sulphonate.



Sulpharsenobenzene is a yellow powder, soluble in water giving a faintly acid solution, and containing about 20 per cent. of arsenic. It is less irritant than salvarsan, and can be given hypodermically, intramuscularly, or intravenously. It resembles neosalvarsan in its actions, but is less active therapeutically, and is stated to be less toxic in that it causes fewer immediate reactions, though the incidence of late reactions is high.

Bismarsen (Sulpharsphenamine Bismuth), bismuth arsphenamine sulphonate, is a brownish-yellow amorphous powder, easily soluble in water giving a slightly alkaline solution which slowly oxidises in air. It is used only by intramuscular injection, its toxicity when given intravenously being too great. Its clinical status is uncertain, but it has been used when therapy with other arsenicals was impossible.

Arsenoxide (Mapharsen) *m*-amino-*p*-hydroxy-phenyl arsine oxide, is a fluffy hygroscopic powder which is stable when dry but oxidises on exposure to air and moisture. It is marketed in nitrogen-filled ampoules which also contain sodium carbonate (to produce the sodium salt) and sucrose in amount required to produce an isotonic solution on addition of the prescribed volume of water. It was originally investigated by Ehrlich but pronounced too toxic. Reinvestigation[1] suggests that this is not the case and that its therapeutic activity is about ten times that of salvarsan. No fatality occurred during the first 3,000,000 injections, though the first was reported in 1939.[2]

Toxic Actions of Organic Arsenicals containing Trivalent Arsenic

As has already been pointed out, many of the organic arsenic compounds are unstable and, especially on exposure to air, readily decompose with production of more toxic substances. Hence errors in technique form a potential source of toxic effects. Particular attention must be paid to the following points :—

1. Tubes or ampoules must be tested for leaks, and discarded if leaks are found. The drug must be used immediately the container is opened.

2. Solutions must be made strictly according to the directions. Thus in Toronto two fatalities occurred in one day, and were reported as due to single doses of neoarsphenamine. At the inquest it was proved that the patients had received in error, a concentrated, unneutralised solution of arsphenamine.[3] The distilled water used must be free from the dead bodies of bacteria (neglect of this precaution, which is more important in dealing with salvarsan than neosalvarsan may lead to effects similar to those in protein shock). Great care must be given to the sterilisation of syringes and needles.

3. Solutions must be used immediately, as, on keeping, their toxicity rapidly increases.

4. Solutions designed for intravenous injection must not be allowed to escape into the subcutaneous tissues, or a severe cellulitis may result.

5. Samples of neosalvarsan which do not form a clear solution must be discarded.

A second possible cause of toxic effects is error in manufacture, but danger from this source is now minimised by the stringent regulations under which the substances are tested biologically.

Even when full allowance is made for these causes, there still remain a considerable number of toxic effects. Though many of these are immediately due to the drug, it has to be remembered that the drugs are being administered to unhealthy individuals; that other, independent, pathological conditions may co-exist with the disease actually being treated, and that, either previously or coincidently, other potentially toxic substances (*e.g.*, mercury) may be used and therefore be partly responsible.

Ehrlich pointed out the danger attending the use of salvarsan in cases with syphilitic lesions of heart or brain, and it is now recognised

[1] Tatum and Cooper, *J. Pharm. exp. Ther.*, 1934, *50*, 198.
[2] Simon and Iglaver, *Amer. J. Syph., Gon. and Ven. Dis.*, 1939, *23*, 612.
[3] *J. Amer. Med. Assoc.*, 1921, 76, 5, 7.

that hypersusceptibility is to be expected in the presence of lesions of liver or kidneys, and in a number of general conditions such as Addison's disease. Even in patients apparently organically sound, however, variation in susceptibility undoubtedly occurs. Thus in one case[1] a robust man, thirty-eight years of age, was given 0·5 gram salvarsan intravenously: vomiting and diarrhœa began at once, and he died in convulsions on the fourth day. In another,[2] a single dose of 0·122 gram of neosalvarsan caused a fatal jaundice with *post-mortem* signs indicative of arsenic poisoning; there was, however, a history of previous intensive treatment with mercury.

Early Reactions. Such unpleasant effects as malaise, feeling of nausea or even vomiting, and headache frequently occur during the injection, and though they are by no means universal are sometimes regarded as "normal." There may also be a rise in temperature lasting a few hours, chill and rigor, severe headache, fall in blood-pressure, diarrhœa and albuminuria. These immediate reactions, often followed by œdema and urticaria, are alarming but rarely fatal. They closely resemble the effects of peptone injection, and occur chiefly after intravenous injections involving large volumes of fluid. They are thus less severe and less frequent with neosalvarsan. It follows, of course, that they are particularly liable to occur with severity in asthmatics and patients who show other signs of hypersensitiveness. The danger is lessened by the administration of adrenaline a few minutes before injection of the arsenical.

Later Reactions. The more severe symptoms of poisoning usually occur later than those just considered—and most frequently after the second or third injection.

Skin eruptions may appear at any time up to the eleventh day, may be accompanied by high temperature, and may progress to exfoliative dermatitis. In such cases there is albuminuria, which, in some cases, may develop into nephritis of sufficient severity to be the proximate cause of death. In other cases, death may be due to broncho-pneumonia, following an involvement of the pulmonary mucosa in the skin lesions.

Jaundice may appear at any time from within a few days of the first injection to many weeks after the last administration of an organic arsenical. Early jaundice is rarely fatal. Cases of acute yellow atrophy of the liver have also been reported. It is often doubtful, of course, whether the jaundice is to be ascribed to the drug or to the disease, since jaundice is not uncommon in untreated syphilis. But though the role of arsenic in the causation of the jaundice is debatable, in all cases where jaundice appears, the administration of arsenicals should be suspended.

Nervous lesions form the most frequent cause of death after administration of salvarsan and its analogues. The first symptoms usually appear within two days of the injection. There may be great restlessness, anxiety, and pain in the back of the neck. Muscular twitchings and convulsions are common, and may lead on to coma and death within forty-eight hours. Other symptoms described are cyanosis of the face and extremities, asphyxia, and anuria.

[1] *Med. Klinik.*, Berlin, 1913, 27, 1065.
[2] *New York Med. J.*, 1929, 112, 496.

Post-mortem Findings. In a large number of cases the principal change found after death has been " hæmorrhagic encephalitis," although the condition is much more a thrombosis with consequent œdema or actual hæmorrhage than a genuine inflammation. Changes in the liver, and especially the kidney, would indicate that in certain cases a profound intoxication of a uræmic type has occurred. That many of the fatal cases are very acute instances of arsenical poisoning is borne out by experiments on animals. Heinz showed (1905) that in contradistinction to the usual " gastro-intestinal " form of arsenical poisoning, hyperacute intoxication follows the introduction of arsenic directly into the circulation, the dominant symptom being a paralysis of the nervous system, heart and vasomotor nerves. This " paralytic form," which has been seen, although rarely, in man (Kunkel), shows itself in profound asthenia, fall of temperature, delirium, vomiting and death in a few hours. The findings in cases in which death has been delayed are, in general, similar to those in cases of poisoning by inorganic arsenic.

Analysis. The organic arsenicals of the salvarsan group may be extracted from tissues, etc., by the method of Vitali (p. 402), and estimate in the extract by Lehmann's method (p. 402).

Chemical Tests.

1. H_2S gas gives no precipitate.
2. Reinsch's test for arsenic is positive.
3. Marsh's test is negative in that no metallic mirror is produced. According to Ganassini, neosalvarsan gives a lemon-coloured deposit if the exit tube is heated to a high temperature. The deposit readily dissolves in ammonium hydroxide.
4. With salvarsan solutions, ferric chloride gives a brownish-violet colour which gradually changes to a deep crimson.

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Special Report Series No. 66, Med. Research Council, which gives a complete list of references (1922).

Arsenic in Beer. Towards the end of November 1900, Dr. Reynolds, of Manchester, by keen clinical observation, was led to suspect the presence of arsenic in beer. His suspicions were verified, and led to some very startling revelations in regard to the presence of arsenic in various foods. In his particular cases, however, the arsenic was traced to glucose used in brewing, and then in turn to sulphuric acid used for making glucose. The matter was so important from a national health point of view that a Royal Commission sat upon it. The report of this Commission was issued late in 1903.[1] In the *Lancet* 1903, 2, pp. 1674 and 1746, will be found an instructive summary of this report.

The Commission stated that 6,000, and probably more, people were affected.

[1] Eyre & Spottiswoode, 5½*d.*

The symptoms are fully discussed, and the Commission reports on them thus :—

"There were great differences in individual cases as regards the quantity of arsenic in the beer consumed, the amount of beer taken, and the duration of the period over which arsenical beer was drunk ; and there were also further differences between individual beer-drinkers—*e.g.*, as to age, sex, health, conditions of nutrition, and habits as regards alcohol which determined the extent of their susceptibility to arsenical poisoning at the time when they began to take beer containing arsenic. . . Corresponding to these and other differences, the disease produced by the arsenical beer during the epidemic varied greatly in its manifestations. On the one hand, there occurred throughout the epidemic (and particularly, it would seem, towards its termination, when people had been drinking arsenical beer for many weeks or months, and so had taken considerable quantities of the poison) an abundance of cases in which, once the possibility of arsenic was entertained, there was comparatively little difficulty in deciding on clinical grounds that the illness was consistent with arsenical poisoning. Such cases presented symptoms corresponding to those described as characteristic of subacute poisoning by arsenic, or which are met with in the poisoning which occasionally results from long-continued doses of arsenic taken medicinally. They showed, for example, inflammation of various mucous surfaces, leading to coryza, huskiness, lachrymation, and the like ; gastro-intestinal disturbance and diarrhœa ; peripheral neuritis affecting sensory and motor nerves, and in some cases associated with herpes or with well-marked erythromelalgia, keratosis, or recent pigmentation corresponding to that which not infrequently occurs in persons taking arsenic for long periods.

"On the other hand, symptoms of the above kind were often slight or absent altogether, and one of the most instructive points in connection with the outbreak is the occurrence of cases in which the symptoms, if taken by themselves and apart from the epidemic, would not have appeared to be readily or sufficiently explained by the suggestion that arsenic was the cause of illness. Thus in several comparatively mild cases the sufferers complained merely of burning hands and feet, or they showed a variety of skin eruptions which are observed in many conditions which have nothing to do with arsenical poisoning. In other cases, again, the main symptoms were those resulting from dilated heart ; and special difficulty arose in cases showing evidence of well-marked peripheral neuritis not associated with symptoms pointing clearly to arsenic, and which appeared practically identical with 'alcoholic neuritis,' a disease previously considered to be the result, alike in drinkers of beer and spirits, of the toxic action of alcohol on nerve tissue."

The Commission then deals exhaustively with various articles of diet in which arsenic has been found : glycerine, caramel, baking powders, malt, treacle, jams with glucose, vinegar, Demerara sugar, malted foods.[1]

As this report will doubtless for many years to come be the official standard to which every question on arsenical poisoning will be referred, it is essential that it should be in the hands of every analytical chemist who may be concerned in a case. As we have already stated, it may be obtained from Eyre & Spottiswoode, Parliamentary Printers, London.

In Hyères (1887) an accident with the preparation of wine led to the poisoning of 400 people (Vidal, Marquet et Dubrandy, "Les vins empoisonnés d'Hyères." *Bull. Acad. de Méd.*, 1888).

In February 1932, 300 sailors on French merchant ships suffered from arsenic poisoning which was probably due to the ration wine (in which arsenic was found) being made of grapes sprayed with arsenical insecticides. (Muhlens, *Deut. med. Woch.*, 1932, p. 854.)

Arsenic in Foodstuffs. About the end of the year 1925 several samples of apples from America were found to be contaminated with arsenic due to the spray used to combat the codling moth and other

[1] Cf. *Bostock* v. *Nicholson* [1904] 1 K. B. 725.

parasites. A certain amount of illness was caused, but no deaths or very serious symptoms occurred. Arsenic was found in varying amounts up to 5 grains per pound of apples, and it was practically confined to the skin. With ordinary care the risk of any serious result from the spraying of apple-trees with lead arsenate is infinitesimal.

Chapman[1] has published an interesting paper dealing with the presence of arsenic in crustaceans and shellfish. He found that English oysters examined by him contained from three to ten parts of arsenic per million of the wet substance, while certain samples of Portuguese oysters contained as much as seventy parts per million. Muscles, cockles, whelks, periwinkles, lobsters, crabs, crayfish and prawns all contained arsenic in quantities varying from three parts per million ($\frac{1}{48}$ grain As_2O_3 per pound) in the case of oysters to a maximum of 174 parts per million (1·2 grains per pound) in one sample of prawns. Tinned crustaceans and shellfish were found to be similarly contaminated. Cox[2] discusses the presence of arsenic in fish, especially with reference to the report of the Swedish Commission on arsenic poisoning.[3] Arsenic varying from 0·10 to 3 parts per million was found by the Ramberg method. He reaches the conclusion that the nitric and sulphuric acid digestion process described by Bang and Ramberg gives higher and more accurate results than the older wet combustion method adopted by the Joint Committee of the Society of Public Analysts and the Society of Chemical Industry in 1902.

Bagchi[4] has reported that arsenic was present in many animal foods examined by him in India but particularly in sole, plaice and lobster (2—3 parts per million.)

Toxicity and Fatal Dose. The various compounds just discussed would certainly seem not to have the same degree of toxicity, but they all agree in this, that their effects are always due to arsenic, though the organic preparations are much less poisonous than the inorganic salts. But *vide ante*, p. 401.

The smallest fatal dose hitherto recorded was observed in a case reported by Castle[5]. A woman took half an ounce of Fowler's solution (arsenite of potassium) in unknown doses during a period of five days. She then died, and on examination the stomach and intestines were found inflamed. Death took place by syncope, and there was an absence of vomiting and purging. The quantity of arsenic which here destroyed life could not have been more than *two grains*. In another case, two grains and a half of arsenic, contained in two ounces of fly-water, killed a robust healthy girl, aged nineteen, in thirty-six hours.[6] Hence, in circumstances favourable to the operation of the poison, the fatal dose in an adult **may be assigned at from two to three grains.**

On the other hand there are numbers of cases in which much larger doses have been ingested without fatal effects or indeed in certain cases anything more than gastric discomfort. In Fyfe's cases[7] sausages were

[1] *Analyst*, November 1926.
[2] *Ibid.*, 1925, 50, 3.
[3] *Lancet*, 1923, 2, 531.
[4] Bagchi, K. N., *Indian Med. Gazette* (1941), 76 : 720.
[5] *Prov. Jour.*, June 28th, 1848, p. 347.
[6] *Med. Gaz.*, vol. 39, p. 116.
[7] Fyfe, G. M., *Lancet* (1943), 2 : 614, November 13th.

contaminated with arsenic to the extent of approximately 10 grains of white arsenic per sausage and though 150 people were affected in varying degrees only two died.

Although arsenite of copper is insoluble in water, it is sufficiently soluble in the acid fluids of the stomach to be absorbed into the blood, and apparently two or three grains must be accepted as a fatal dose.

In two cases in 1853, a small quantity of a confectionery ornament, coloured with arsenite of copper, proved fatal to two children. The symptoms and appearances were those of poisoning by arsenious acid. The quantity taken could not have been above two or three grains. The children picked up the ornament in the street and shared it between them. The poison was spread over a layer of sugar.

Orpiment produces symptoms and appearances similar to those caused by arsenious acid; but the dose required to destroy life varies according to the proportion of arsenious acid with which it happens to be mixed, the acid being more toxic than the orpiment.

Duration. Arsenic is an irritant to the stomach, even in small quantities. It appears to have a specific irritant effect which occurs only after absorption into the cells; it is therefore different from the corrosives and many other irritants. It has also definite toxic effects on cellular structures, especially on the cells of the kidney and liver and muscle of the heart. The time of onset of symptoms depends on the rate of absorption of the arsenic, which depends on the physical state of the ingested material (solution, fine or coarse powder, etc.) as well as its exact chemical nature, and on the nature and amount of food in the stomach. Symptoms in **acute cases** usually come on within a quarter to half an hour after the dose, and sometimes even earlier. On the other hand, in an instance where one drachm had been taken on an empty stomach, no symptoms appeared for two hours. In a case reported by Orfila, the symptoms did not show themselves for five hours; and in another, in which a large dose was taken, the symptoms did not come on for seven hours. There may be every variety between these extremes. In one case their appearance was postponed for *ten* hours. A remarkable instance occurred in which the poison was taken by a young female at eleven o'clock in the morning, and no well-marked symptoms occurred for *eight hours*: there was then violent vomiting. After death, a cyst, formed of mucous membrane containing arsenic, was found in the stomach, the poison having thus become sheathed over.[1] In another case,[2] symptoms of violent irritation did not show themselves until twenty-three hours after the poison had been taken, and within about half an hour of the death of the patient. The girl was sick once shortly after having taken the poison, but the first symptoms were those of narcotism. She was a confirmed opium-eater, and this habit may have had some influence in delaying the operation of the poison. In all cases in which arsenic enters the system from without, as by its application to the skin, or to ulcerated or diseased surfaces, the symptoms are delayed. Large doses of arsenic commonly prove fatal in from eighteen hours to three days. The average time at which death takes place is twenty-four hours; but the poison may destroy life within a much shorter period. There are many authentic cases reported in which death has occurred in from three

[1] Flandin, vol. 1, p. 535.
[2] *Med. Times*, October 21st, 1848.

to six hours. Taylor met with a case of death from arsenic in five hours ; and in another, in two and a half hours.[1] Foster met with the case of a child under three years of age which died within *two hours* from the effects of arsenic. The quantity taken could not be determined. As a general rule, children die much more rapidly than adults, and we have seen a number of cases in which death occurred within four hours. In some instances death does not occur until long after the average period. In one case in which an adult swallowed about half an ounce, death did not take place for *fifty hours*, and it is remarkable that there was an entire absence of pain.[2] It is obvious that a patient who recovers from the first effects of this poison may still die from exhaustion or other secondary causes many days or weeks after having taken it, even although the whole of the poison has been eliminated from the body.

Symptoms. In a **case of acute arsenical poisoning** by the mouth the individual usually first experiences faintness, depression, nausea, and sickness. There may be a dry or burning feeling about the mouth, followed by salivation. An intense burning pain is felt in the region of the stomach, increased by pressure. The pain in the abdomen becomes more and more severe ; and there is violent vomiting of brown turbid matter, mixed with mucus, and sometimes streaked with blood. These symptoms are followed by purging, which is more or less violent, and this may be accompanied by severe cramps in the calves of the legs ; these are, however, often absent. The matters discharged from the stomach and bowels have had in some instances a yellowish colour, due, it was supposed, to a partial conversion of the poison into sulphide, but more probably due to an admixture of bile. The vomited matters are in some cases coloured with blood, and the mixture of blood with bile has often given to them a green or brown colour. In other cases, they may consist of a large quantity of mucus ejected in a flaky state and having a milky-white appearance, as if from admixture with the poison. The colour of the vomited matters may be blue or black when coloured arsenic has been taken, or the admixture of bile may render them a deep green. The vomiting may be violent and incessant, and excited by any liquid or solid taken into the stomach ; in many cases, however, there are remissions during which the patient is moderately comfortable. There is tenesmus (straining at stool), and the discharges by the bowels are frequently tinged with blood. There is a sense of constriction, with a feeling of burning heat in the throat, commonly accompanied by the most intense thirst. The pulse is small, frequent, and irregular, sometimes wholly imperceptible. This is almost constant and is probably due to the direct action of the poison on the heart muscle. The skin is cold and clammy in the stage of collapse ; at other times it is hot. The respiration is painful from the tender state of the stomach. There may be redness and irritation of the throat and conjunctivæ. The patient lies in a state of exhaustion with pinched and sunken face, due to the excessive loss of body fluids. There is great restlessness, but before death stupor may supervene, with paralysis, tetanic convulsions, or spasms in the muscles of the extremities. Although pain is in general among the early and well-marked symptoms, arsenic appears in some cases to destroy

[1] Guy's Hosp. Rep., 1850, p. 183 ; see also " Ann. d'Hyg.," 1837, 1, 339.
[2] *Med. Gaz.*, vol. 48, p. 446.

sensibility. Thus it has been observed that, even when the stomach has been found intensely inflamed after death, the patient had not complained of pain during the time which he survived.

It is not, however, in every case that all these symptoms are observed. Cramps in the calves may be absent; and there may be no vomiting or purging. In one case, where a woman died in three hours after taking arsenic in a pudding served at dinner, there was no vomiting or purging. In two hours she was in a state of complete collapse, and at the time it was noticed that the conjunctivæ were red.[1] Vomiting and purging are nevertheless seldom both absent. The condition of the urine has been stressed; but as it has been found normal, retained, suppressed, or abundant, no clinical importance can be attached to the increase or diminution of this secretion. The condition of the urine was much discussed in *R.* v. *Maybrick*.[2]

In October 1891, a gamekeeper drank an unknown quantity of wine, accidentally contaminated with from five to six grains of arsenious acid per fluid ounce; and subsequently four other members of the family partook of the wine, in quantities varying from a mere taste in the case of a young child to half a tumblerful or more in the cases of the adults.

The man suffered from severe abdominal pains, persistent vomiting, and diarrhœa, and cramps in the calves of the legs, the feet and the hands. He died in two days. The child suffered somewhat from the effects of the irritant. The other three adults (young women), in about ten minutes, all had a burning sensation in the throat and gullet, vomiting, diarrhœa, abdominal pains, but no cramp in the limbs. On *post-mortem* examination of the deceased man, the signs of acute gastro-enteritis were visible, but none of the petechial ecchymosis of the mucous membrane of the stomach, which has by some observers been regarded as characteristic of arsenical gastro-enteritis.

Chronic Poisoning. In the majority of accidental cases, and in many homicidal cases, the symptoms are not of this severe and definitely gastro-intestinal character. In these cases, which may be termed chronic to distinguish them, the symptoms may be put into four groups: —

(1) Gastro-intestinal symptoms of modified severity.
(2) Catarrhal symptoms about the eyes, nose and mouth.
(3) Cutaneous rashes, with discolorations.
(4) Symptoms of peripheral neuritis.

Should the person recover from the first effects, and the case be protracted, or should the dose have been small and administered at intervals, there will be a general feeling of malaise and want of energy, associated with loss of appetite and attacks of vomiting and diarrhœa. Inflammation of the conjunctivæ, with suffusion of the eyes, and intolerance of light occur, conditions which are, however, often present among the early symptoms above described. Catarrh of other mucous surfaces, nose, pharynx, larynx, trachea, etc., is common. There is also irritation of the skin, accompanied by a vesicular eruption, which has been called *eczema arsenicale*. Sometimes this has assumed the form of nettlerash or of the eruption attending scarlet fever, for which disease arsenical poisoning has been mistaken. There may be a patchy brown discoloration of the skin, scaling, thickening, or keratosis of the palms and soles. Symptoms referable to the central nervous system are characteristic of

[1] *Med. Times*, 1851, 2, p. 229.
[2] Liverpool Sum. Ass., 1889.

chronic arsenical poisoning. Tingling and numbness of the hands and feet and tenderness of the muscles are often observed in about ten days, followed by paresis or paralysis. The paralysis affects the extensors of the feet, then the hand and later the muscles of the calf and forearms. Sir Thomas Stevenson noted a case of arsenical poisoning in which repeated three-quarter grain doses of white arsenic given with homicidal intent were followed by general paralysis beginning in the lower extremities, and gradually creeping upwards till the lower intercostal and other respiratory muscles were affected. The patient recovered under treatment. Exfoliation of the cuticle and skin of the tongue, herpes, and falling off of the hair, have likewise been witnessed. Salivation has been observed to follow, especially when small doses of the poison have been given for a length of time. Strangury, albuminuria and jaundice have also been noticed among the secondary symptoms from the toxic action on the kidneys and liver. Fatty degeneration of the heart and vessels may occur.

At Hyères in 1887 an alarming outbreak of arsenical poisoning occurred, the poison having been accidentally introduced into wine, which was drunk by many persons for a considerable time. The symptoms at the outset were those of dyspepsia, with nausea, vomiting, and purging; but nausea and vomiting were not invariable. The gastro-intestinal symptoms generally disappeared in a few days. There was dryness of the mouth, loss of appetite, a sense of constriction at the pit of the stomach, and wasting. Bronchial irritation was marked, with scanty secretion of mucus. There were pains in the limbs and œdema of the joints. The patellar reflexes were weakened or abolished. Wandering pains were felt in various regions, and headache, and the sense of touch was diminished. A garlicky taste was perceived in the mouth, but the sense of taste was not diminished. The vision was disturbed, and the conjunctiva of the eye was inflamed. There were scaly eruptions on both surfaces of the hands and feet. Of sixty-three reported cases, thirty were slight, eighteen serious, and fifteen fatal.

In any one group of these accidental cases the *symptoms* produced are of a more or less uniform character, showing their origin from a common cause. In the cases derived from wall-papers they were as follows:—dryness and irritation of the throat, dry cough, irritation of the mucous membrane of the eyes and nostrils, languor, headache, loss of appetite, nausea, colicky pains, numbness, cramp, irritability of the bowels attended with mucous discharges, great prostration of strength, sleeplessness, a feverish condition, and wasting of the body. These symptoms may not all have presented themselves in any one case. The connection of the symptoms with this cause appears to have been in some instances clearly established by the fact that after the removal of the paper, especially from bedrooms, the symptoms have disappeared.[1]

Ferrannini,[2] after discussing the ordinary symptoms of poisoning by arsenic, refers briefly to some of the rarer forms:—

"In addition to paralysis one may get ataxia, which may be associated with other tabetic symptoms—for example, absence of knee-jerks, Romberg's sign, lightning pains, anæsthesia, and ocular disorders (diplopia, amaurosis, absence of pupillary reflex). This ataxic form is due to a polyneuritis, and not a myelitis.

[1] *Med. Times and Gaz.*, 1871, 1, p. 67.
[2] *Rif. Med.*, June 3rd, 1900 (*B.M.J.*, Epitome, July 18th, 1903).

Tremor may also be due to arsenical poisoning. In differentiating between alcoholic and arsenical poisoning, the following data should be borne in mind. In the first place alcoholic neuritis is rarely due to acute alcohol poisoning, but usually occurs in the chronic drinker, and the delirium which opens the scene is the equivalent of the acute gastro-enteritis of arsenical poisoning. In alcoholic dyspepsia chronic gastritis with morning catarrh and pyrosis are the common type. In arsenicism you get insomnia, in alcoholism terrifying dreams. The sensory disturbances in alcoholic paralysis are usually less severe than in arsenical poisoning. Desquamation is peculiar to arsenical poisoning, whilst psychical disorders prevail in alcoholism. In arsenical poisoning the motor disorders, the atrophy and deformities chiefly affect the fingers and toes ; in alcoholic poisoning the forearm and calf are more prominently affected, and it is the wrist and ankle articulations rather than the phalangeal which share in the deformity. The author then relates three cases of arsenical paralysis occurring in the same family (mother and two sons), and presenting identical symptoms, due to eating flour made from lathyrus beans (vetch) into which arsenic had been put. In each case it was the lower extremities that were paralysed. Sensory disturbances (itching, numbness) were present in the palms of the hands and soles of the feet. Some static ataxia was present. The tendon reflexes were abolished. Sensation normal. There was considerable wasting of the forearm and leg."

Treatment. In acute cases the stomach must be washed out frequently with warm water by means of the stomach tube and *freshly precipitated ferric oxide* administered. This can be prepared by adding ammonia water, or a solution of potassium carbonate, to the tincture of iron perchloride ; the precipitate is strained off and administered suspended in water. Calcined magnesia or charcoal may be substituted if ferric oxide cannot be obtained. Demulcents and subsequently morphine should be given. For thirst, ice may be given to suck. External warmth will be required. Careful attention must be directed to the circulatory system and the heart suitably supported. Intravenous injections of warm saline with 5 per cent. glucose should be used to combat shock, and the addition of alkali is of use in the majority of cases.

Treatment by Sodium Thiosulphate. Revaut[1] first suggested the use of this salt for the treatment of poisoning by metals. The subject was further investigated by Dennie and McBride,[2] by Semon[3] and many others.

It is used intravenously in doses of 0·3–0·6 grm. in 10 per cent. solution daily until improvement occurs. In the dermatitis produced by organic arsenic preparations, there is now a considerable volume of evidence to show that it has a favourable influence. It has also been alleged to give good results in poisoning by the ordinary arsenical salts and by preparations of mercury, lead and bismuth ; but the evidence for the latter is by no means so good. Although Groehl and Meyers[4] state that it hastens the excretion of metals, oral administration of the salt has no apparent value.

Treatment by Sulphydryl Compounds.

An important advance was made when the theory was evolved that the point of attack selected by arsenic after absorption, and conversion into the trivalent form if necessary, was the sulphydryl (SH) compounds

[1] *Presse Médicale*, January 28th, 1923.

[2] *Arch. Derm. and Syph.*, January 1923 ; *Jour. Amer. Med. Assoc.*, December 27th, 1924.

[3] *B.M.J.*, April 12th, 1924.

[4] *Therap. Gaz.*, October 15th, 1924.

of the tissue cells—which acted, in fact, as arseno-receptors. That some such explanation was feasible, or even probable, was favoured by the previously observed fact that arsenic after absorption could be recovered in very considerable concentration from epidermal (keratin) tissues known to be rich in sulphur (in the sulphydryl form).

A logical development was the attempt to use a suitable SH compound in the prevention or treatment of arsenical intoxication. Glutathione (a monothiol) was chiefly used by Voegtlin and others[1]—and it was found that injections of glutathione did have an inhibiting effect so far as the poisonous results of arsenic were concerned. (The sulphur in such compounds bears no relationship to the sulphur in thiosulphate, and Voegtlin's work gives no experimental support for the use of the latter substance.) But the glutathione had to be administered very soon (within minutes) after the arsenic, and it had to be given in large quantities. Even then the protection afforded was incomplete, and was not effective against all arsenical compounds. Nevertheless, the work represented a great step forward in the understanding of the effects of arsenic and in an approach to a rational treatment of arsenical intoxication by a real physiological antidote.

About 1936, Peters and others were investigating the action of certain non-arsenical vesicants, which were found to have a selective action on the carbohydrate (pyruvate) enzyme system in brain tissue. During their investigations, they found that sodium arsenite—a non-vesicant—had a similar effect. With the advent of war, they were called upon to devise, if possible, an antidote to Lewisite, which is an arsenical poison having both vesicant and general effects. An early step was therefore to test the sensitivity of the pyruvate oxidaze system to arsenicals, a test which showed (*a*) that the pyruvate oxidaze system was specially sensitive to arsenicals; and (*b*) that the attack was primarily upon an SH-containing protein component of the system. This was consistent with the previously held theory, but represented a distinct advance in that it defined more precisely the mode of action of arsenic as a poison. Arsenic is enzyme selective. It is not a protein precipitant. It acts as a poison by virtue of its effect on the function of the living cell, not on its structure.

Further work showed that monothiol compounds such as glutathione were not, in general, really effective in protecting the tissues against the action of Lewisite or arsenite. Indeed they might themselves have pronounced toxic effects. But in studying the manner in which the arsenic combined with the pyruvate oxidaze system, it was noted that most of the arsenic was combined in the proportions of 1As : 2 SH, probably with 2 SH groups close together on the same molecule to form a ring compound. It seemed probable, therefore, that greater protection might be given by a dithiol compound, capable of forming a stable ring compound with arsenic, rather than by the open chain, monothiol compounds previously investigated.

Various dithiol compounds were tried, and success was achieved with the compound which is now known as B.A.L. whose chemical composition is indicated by the name 2 : 3 Mercaptopropanol. This substance was found to be capable of preventing the action of Lewisite : of preventing

[1] *Jour. Pharmacol. and Exp. Therap.*, May 1925.

the serious effects of Lewisite even after a delay of an hour or more; and, most significant of all, of effecting a very substantial reversal of the effects of Lewisite when these had become established. B.A.L. is apparently able to withdraw the arsenic from its union with the SH radicles in the tissue enzyme. These effects are seen not only in regard to the vesicant effects of Lewisite, but also in respect of its general effects, and of the general effects of arsenite poisoning. In all cases, the administration of B.A.L. is rapidly followed by a great increase in the excretion of arsenic in the urine.

B.A.L. was first prepared as a penetrating ointment, in which form it could be used both prophylactically and therapeutically. This form has now been superseded by a solution of B.A.L. in 10 per cent. benzyl benzoate in peanut (arachis) oil. The B.A.L. content of the solution is either 5 per cent. or 10 per cent. The therapeutic use of such B.A.L. solutions has so far been chiefly in cases of arsenical dermatitis or other complications of arseno-therapy or conditions due to industrial contact with arsenical dusts. The experimental work, however, favours a belief that B.A.L. must now be recognised as a form of treatment which holds out every prospect of success in poisoning with even large doses of inorganic arsenic by mouth. Impressive clinical results have already been reported in the treatment with B.A.L. of cases of arsenical dermatitis, encephalitis, blood dyscrasias "arsenical fever", and accidental massive overdosage with organic arsenicals. The results in cases of post-arsenical jaundice are more doubtful, though some cases have responded well. This latter finding is in keeping with the doubts which have been expressed as to the actual role of arsenic in the production of jaundice.

The suggested dosage for a severe case of arsenical poisoning is 3 mgm/kilo of body weight given by intramuscular injection every four hours for the first two days, and twice daily thereafter for ten days or until recovery is complete. For milder cases, the dose may be smaller, *e.g.* 2·5 mgm/kilo, and the frequency of the injections need not be so great. From the recorded experience of the use of B.A.L. in mercurial poisoning it appears reasonable to think that in cases of acute poisoning by ingested inorganic arsenic in considerable quantity, still more intensive therapy may be advisable. (See section on mercury poisoning, p. 366.) High dosages of B.A.L. may cause toxic symptoms, some of which may resemble the effects of the arsenic itself, *e.g.* nausea, vomiting, lacrymation, tingling of the extremities, etc. But when one considers the desperate plight of victims of acute arsenic poisoning, desperate measures are justifiable.

As previously indicated, B.A.L. has also been used in the treatment of poisoning by other metals, notably mercury and gold. The effects of such treatment indicate that the "dithiol theory" of action applies also to these two metals, and possibly to others, *e.g.* copper and zinc. There is no doubt therefore, that in B.A.L. we now have a powerful therapeutic agent for use in cases of poisoning by arsenic, inorganic or organic, acute or chronic; and by mercury, and gold, and possibly other metals as well. It is important that B.A.L. therapy should be adequate as regards dosage, and that it should be started as early as possible. The greater the delay in instituting treatment, the less will be the preventive action, and the lesser the extent of the reversal which B.A.L. can accomplish. If the arsenic or other metal inactivates the tissue enzymes for a sufficiently long period, then the damage done to the cells will be irreversible, even

if the B.A.L. should succeed in withdrawing the arsenic from its tissue SH combination. Early treatment with B.A.L. is therefore of prime importance. Although B.A.L. can cause toxic symptoms, it appears that there are few real contra-indications to its use, except perhaps the existence of liver damage. Kidney damage is not a contra-indication. Indeed, it may be said that in severe, and therefore desperate, cases of arsenic or other metal poisoning, B.A.L. should in all circumstances be given, if it is available. The use of B.A.L. does not obviate the necessity for the other general measures which are recognised as being of value in cases of poisoning in general, and the metallic poisonings in particular. But the addition of B.A.L. to the therapeutic armamentarium has improved the prospects of success with which such cases can now be treated.[1] [2]

Post-mortem Appearances. The striking changes produced by arsenic are generally confined in the acute cases to the stomach and bowels. They are commonly well marked in proportion to the largeness of the dose, and the length of time which the person has survived after taking the poison. Our attention must be first directed to the *stomach*. Arsenic seems to have a specific effect on this organ, and the mucous membranes generally : for, however the poison may have entered into the system, whether through a wounded, diseased, or ulcerated surface, or by the act of swallowing, the stomach has been found inflamed. The mucous membrane of the stomach, which is often covered with a layer of mucus, mixed with blood or bile, and with a thick, white, pasty-looking substance containing arsenic, is commonly found red and inflamed in dotted or striated patches ; the colour, which is of a dull or brownish red, becomes brighter on exposure to the air : at other times it is a deep crimson hue, interspersed with black-looking lines or patches of altered blood. The redness is usually marked most strongly at the greater end, but may be found spread over the whole mucous surface, giving to it the appearance of red velvet ; in other cases it will be chiefly seen on the prominences or folds of the membrane. Often there is punctate ecchymosis, though this is not invariable. The mucous coat may be thickened and of a gelatinous consistency, without any marked inflammatory redness.

The stomach has been found highly inflamed in a case which proved fatal in *two hours*. Thus it would appear that inflammation of the mucous membrane may be well marked within a very short period.

A woman, *æt.* 24, retired to her bedroom after dinner at two o'clock. At three o'clock she was not suffering from any apparent illness. At half-past four she called her sister, and then it was found that she had swallowed a quantity of arsenic. There was then no sickness. After this, she was sick once, and purged once, but complained of no pain. She drank some tea, but almost immediately became collapsed, and seemed to those who were with her to be falling into a fainting fit. She died before six o'clock, and was sensible to the last. She could not have taken the poison more than two hours before she died.

On inspection the day following, the whole mucous membrane of the stomach was intensely inflamed, presenting a dark scarlet colour, with broad livid patches. Upwards of one ounce of solid arsenic was found in a pasty state on the mucous membrane of the stomach, which was raised, thickened, and velvety.

[1] Peters, R. A., Stocken, L. A., and Thompson, R. H. S. : *Nature*, 1945, 156, 616.
[2] *Biochem. J.* 1947, 41, 53.

This case shows not only that there may be extensive morbid changes in the body within a short period after the taking of the poison, but that, with an unusually large dose, the symptoms of vomiting, purging, and pain may be slight and bear no proportion to the quantity of poison taken. Blood of a dark colour may be effused in various parts within the folds, or beneath the lining membrane. A raised circular or oval patch of false membrane with an intensely red border, and with arsenic upon its surface, may sometimes be seen upon the inner coat.

The stomach often contains a mucous liquid of a dark colour tinged with blood. The coats are sometimes thickened in patches, being raised up into a sort of fungus-like tumour, with arsenic embedded in them : at other times they have been found thinned. The mucous membrane may be found ulcerated, and still more rarely gangrenous. Ulceration of the mucous membrane, as the result of the action of arsenic, has been found as early as ten hours after the poison had been taken. Perforation of the coats is so uncommon a result of arsenical poisoning, that there are but few instances on record. Murray Thompson met with a case in which there was on the mucous coat of the stomach a black hardened patch the size of the palm of the hand.

Arsenic may be seen deposited as the yellow sulphide under the peritoneal coat of the stomach, especially in cases where putrefaction has commenced. White arsenic may be so completely converted into the sulphide that in certain cases the Reinsch test cannot be used.

In a few instances the mouth, throat, and gullet have been found inflamed, but in general there are no changes in these parts to attract particular attention. The mucous membrane of the small intestines may be inflamed throughout, but commonly the inflammatory redness is confined to the upper part or to the duodenum, especially to that portion which joins the stomach. Of the large intestines, the rectum appears to be the most prone to inflammation.

It is worthy of observation in relation to the known antiseptic properties of arsenic, that the parts especially affected by the poison (the stomach and bowels) occasionally retain the well-marked characters of irritant poisoning for a long time after death. Absorbed arsenic does not, however, appear to prevent markedly the decomposition of the soft organs in which it is deposited.

So long as the case is acute the exact method of entrance, or rather portal, of the poison seems to have very little influence in preventing the stomach from being attacked. Thus in some cases in which baby's powder contaminated with arsenic was applied to the skin, arsenic was found in the stomach and contents, and its presence there might have led to an erroneous inference of its having been criminally administered by the mouth. It was, however, present merely in traces, and obviously the result of elimination. The nature and mode of occurrence of the symptoms were also opposed to any other presumption. That absorbed arsenic may be thus transferred from the blood to the stomach and intestines, has been distinctly proved by the experiments of Pavy and Taylor.[1]

Another female infant died fourteen days after birth. In this case a similar violet powder had been used. Within a short time of the

[1] Guy's Hosp. Rep., 1860, p. 397.

application the skin became red, and vomiting and purging set in, and continued till death. On section the abdominal parietes showed much inflammatory action ; they were thickened and adherent to the viscera. The rectum was highly inflamed ; the kidneys and spleen were much congested ; and the liver was very slightly congested. Upwards of three grains of arsenic were extracted from the viscera.

Of course, if some of the coloured arsenical compounds have been administered, it is possible that there may be coloured particles visible in the alimentary canal.

Small hæmorrhages are frequently found in the wall of the ventricles of the heart under the endocardium and capillary hæmorrhages may be present elsewhere. Changes in the kidneys and liver, as the result of absorption of arsenic, are not visible to the naked eye, but microscopic examination shows evidence of cloudy swelling and fatty degeneration. These changes are also observed in the heart muscle and in the mucous membrane of the stomach. The *post-mortem* findings may be summed up as follows :—The alimentary organs and heart show characteristic changes, and if these be absent then the effects of arsenic can only be proved by analysis, and not even by that alone in all cases without the clinical symptoms, for arsenic is rapidly eliminated and may not be found even when we are sure death was due to it.

Distribution in and Elimination of Arsenic from the Body. Arsenic after absorption from the intestinal canal passes to the liver, where a sensible proportion is stored. From the liver it passes to the whole of the body by the blood-stream and is excreted in great part by the kidneys, but to a certain extent by the alimentary canal.

After a single dose arsenic is found in the urine within half an hour, and continues to be excreted for about ten to fourteen days. In acute arsenic poisoning it is most exceptional to find the metal in the urine for a longer period. After repeated doses of arsenobenzol it may be found for much longer periods.

The elimination of arsenic by means of the intestinal canal takes place for a similar period.

Ingested arsenic after absorption is found in greatest quantity in the liver. The kidneys contain proportionately less, and the other organs are likely to contain traces according to their blood-content.

Van der Rieb, Copeman and Kamerman,[1] report the following figures from six of their cases.

Arsenic (As_2O_3) in mg. per 100 g.			Arsenic (As_2O_3) in mg. per total organ.		
Liver.	Kidneys.	Stomach.	Liver.	Kidneys.	Stomach.
0·9	0·6	0·4	30·0	2·6	3·9
4·8	2·1	30·0	77·0	6·5	143·0
6·0	1·8	1·2	65·0	7·1	4·5
15·0	13·1	225·0	120·0	17·5	227·0
1·3	0·8	0·2	13·7	1·3	0·7
3·3	1·5	1·1	27·3	3·2	3·2

They point out that the ratio between the concentrations in the liver and kidney is very variable (average about 2) as is the ratio between the

1 Chemistry Series No. 164. Government Chemical Laboratory, Johannesburg, 1941.

total amounts (average about 10). Nevertheless, their results support the statement that the kidneys contain less arsenic per 100 g. than does the liver.

Arsenic, especially in prolonged administration, is likely to be found in the hair, epidermis, nails, and bones. A series of experiments carried out by one of the editors (S.S.) has shown that arsenic is readily absorbed by keratin tissues in general either from the body fluids *via* the living cells or by contamination from without. The concentration in hair and nails thus contaminated is likely to be much greater than the concentration of arsenic in the contaminating fluid.

The distribution of arsenic in the viscera and keratin tissues and the relationship between this amount and the amounts found in the alimentary canal offer a means of ascertaining the probable time or times of administration, although no precise opinion can be given in the majority of cases. Hair grows at the rate of about half an inch per month, and an examination of successive small lengths of hair from the root upwards may give some indication of the length of time that has elapsed since arsenic was given to the individual.

Willcox[1] gives the distribution of arsenic in the organs of the victims in certain cases tried in the English courts, which are most informative in this respect.

Maybrick Case

	Grains.	Milligrams.
Stomach	*nil*	*nil*
Intestines	$\frac{1}{11}$	6
Kidney	trace	trace
Liver	0·3	21
Total	43/100	27

Rex v. *Bingham* (*Re Margaret Bingham, deceased*)

Death in 3 or 4 days from onset

	Grains.
Stomach	$\frac{1}{250}$
Liver	1·1
Kidneys	$\frac{1}{130}$
Spleen	trace

Rex v. *Bingham* (*Re James Bingham, deceased*)

Onset, August 12th, 1911 (3 p.m.)

Death, August 15th, 1911 (evening)

	Grains.
Stomach	minute traces
Liver	$\frac{1}{6}$
Kidneys	$\frac{1}{25}$
Spleen	$\frac{1}{300}$

[1] *Trans. Med.-Leg. Soc.*, vol. 16, p. 185.

Rex v. *Bingham* (*Re William Hodgson Bingham, deceased*)

Onset, January 22nd.
Death, January 24th (early morning).
Duration, ? 36 hours, about.

	Grains.
Stomach	$\frac{1}{12}$
Small intestines	$\frac{1}{40}$
Liver	1
Kidneys	$\frac{1}{250}$
Spleen	$\frac{1}{500}$

Seddon Case

	Grains.	Milligrams.
Stomach	0·11	7·30
Intestines	0·63	41·00
Liver	0·17	11·13
Kidneys	0·03	1·91
Spleen	0·007	0·44
Lungs	0·014	0·94
Heart	0·012	0·80
Brain	0·005	0·33
Blood (fluid from chest)	0·006	0·38
Bone	trace	trace
Nails	0·002	0·14
Skin (0·18 mgrm. per 100 grms.) ..	—	—
Muscle	1·03	67·20
Hair (proximal, 3 mgrms. per 100 grms.)	—	—
Total	2·01	131·57

Rex v. *Greenwood* (*Re Mrs. Greenwood, deceased*)

	Grains.	Milligrams.
Stomach	0·009	0·6
Small intestines	0·067	4·33
Large intestines	0·008	0·55
Rectum	0·006	0·40
Liver	0·131	8·5
Spleen	0·006	0·4
Kidneys	0·019	1·21
Uterus	0·012	0·75
Heart	0·004	0·27
Lungs	0·012	0·79
(Esophagus	0·003	0·21
Brain	0·001	0·09
Total	0·28	18·07

Rex v. *Armstrong* (*Re Mrs. Armstrong, deceased*)

	Milligrams.
Stomach	2·5
Stomach contents	2·0
Jejunum and contents	1·6
Ileum and contents	9·1
Cæcum, ascending colon, and contents	37·6
Liver	138·0
Spleen	1·0
Kidneys	13·2
Left lung	0·5
Heart	0·6
Fluid from pleural cavities	0·9
Portion of brain (14 oz.)	0·1
Bone from left femur (4 oz.)	0·01
Skin from left thigh ($3\frac{1}{4}$ oz.)	0·25
Muscle, back of left thigh ($3\frac{1}{2}$ oz.)	0·21
Hair from head ($3\frac{3}{4}$ oz.)	0·54
Finger-nails	0·06
Toe-nails	0·03
Total	208·2

In making any deduction as to the time when the last dose of the poison was taken, the greatest care must be exercised, and it must be remembered that arsenic occasionally remains in the stomach for at least twenty-four hours, even though vomiting has taken place and stomach lavage has been applied.

In the case of *Rex* v. *Hearne* (Bodmin Assizes, June 1931), the body of Miss Everard, the sister of the accused, was exhumed five months after death, and arsenic was found in the viscera and keratin tissues in the following amounts :—

Organ.	Arsenic. Parts per million.
Stomach	1·3
Small intestine	1
Large intestine	5
Liver	2
Bone	0·7
Muscle	3
Skin	2·7
Nails	40

A lock of hair $4\frac{1}{2}$ inches long was divided into three portions and separately analysed with the following results :—

Scalp end	23
Middle portion	15
Free end	10

The analyst was of opinion that this distribution indicated that arsenic had been given to the deceased at least seven months before her

death and that towards the end the doses had been increased. The distribution in the viscera and keratin tissues tends to support that opinion.

The soil in which the body was buried, however, was found to contain arsenic, and the defence drew attention to the possibility of contamination of the body by water percolating through the soil and penetrating the coffin. Certain experiments carried out by one of us (S.S.) with reference to the absorption of arsenic by the keratin tissues indicated that nails and hair, and particularly the former, had the capacity of absorbing large quantities of arsenic from relatively weak solutions. The excessive amount of arsenic in the nails and hair therefore might have been due to contamination from without.

The other victim in the case, Mrs. Thomas, was taken ill on October 18th with symptoms suggesting that a toxic dose of arsenic was administered on that day. Acute symptoms continued for several days and on the 24th tingling of the feet was observed and on the 29th herpes of the lip; on the 30th weakness of the legs was observed and on November 3rd the condition became more serious; on the 4th she died. Seventeen days elapsed between the ingestion of the poison and death.

An examination of the viscera showed the following distribution:—

Organ.		Total Quantity Found.
Stomach	6·7 ozs.	·0077 of a grain
Intestines	62 oz.	·069 ,, ,,
Liver	49 ozs.	·66 ,, ,,
Kidneys	16 ozs.	·12 ,, ,,
Hair ..	.. ..	7 parts per million.

The quantity found in the liver after the length of time which was supposed to have elapsed appears excessive and leads to the suggestion that poison had been absorbed at some period between the onset of the symptoms and death.

The amount present in the hair, 7 parts per million, has a certain interest, but unfortunately the whole lock of hair was examined in one lot. Since it is only possible for the hair adjoining the scalp to have been impregnated by arsenic from the hair root, an examination of the whole length of the hair gives no information of value. Furthermore, it was ascertained during the trial that the hair had been removed after the *post-mortem* examination had been completed and therefore the possibility of its being contaminated with blood or body fluids must be borne in mind.

The excretion of arsenic is mainly in the urine. Experimental intravenous injection of various arsenicalsinto rats, by Voegtlin and Thompson and others,[1,2] indicates that, while the excretion rate varies from individual to individual, it is most rapid in the case of the pentavalent compounds, and slower in the case of the trivalent arsenious acid. The toxicity of the trivalent preparations is greater than that of the pentavalent, and it would appear that the toxicity of arsenicals is in a sense determined by their rate of excretion. After injection, arsenicals leave the blood-stream rapidly, the trivalent forms probably becoming fixed in the

[1] *Journ. Pharm. and Exp. Therap.*, 20 : 85-105, 1922-23.
[2] *Ibid.*, 20 : 129, 1922-23.

tissues, while the pentavalent forms are to a large extent excreted before becoming fixed. Probably the pentavalent forms are reduced to the trivalent before they exert a marked toxic effect, and the trivalent forms must be re-oxidised to the pentavalent before excretion. In rats, the toxicity of the arsenite is about seven times as great as that of the arsenate, while the excretion rate of arsenate is two and a half times higher than that of the arsenite. The percentage of the dose recovered after intravenous injection of various arsenicals is given as follows :—

	In 6 hours. Urine.	In 18 hours. Urine.	In 20 hours. Urine.	In 20 hours. Fæces.	In 24 hours. Urine and Fæces.
Arsenious Acid	12·7	·69	13·4	4·7	19·8
Arsphenamine	6·05	5·1	11·2	82·3	93·5
Neo-arsphenamine ..	34·4	19·7	52	33·3	87·6
Arsenic Acid	45·6	5·3	51	2	53
Amino-phenyl Arsenic Acid	86·3	8·6	93	5·6	97·97

It will be observed that the excretion of arsenic into the intestines is negligible, except in the case of arsphenamine, where 82 per cent. of the injected arsenic was excreted by the bowel within 24 hours, and to a lesser extent in the case of neo-arsphenamine.

Analysis. General Considerations. Owing to the fact that arsenic is one of the few poisons which have been used in historical cases of chronic poisoning, a halo of romance and mystery has been associated with this substance which led Taylor to record everything reported on known cases. We are now in a much better position to collate and explain much that was to Taylor mysterious and almost supernatural, and even to reconcile many points in apparent conflict in his estimation.

Thus Taylor thought it peculiar that a person might die of arsenic and yet none be found in the body. We now know that arsenic is **not an accumulative poison** ;[1] it is temporarily deposited in the organs after absorption, but is eliminated by the urine and other secretions ; and in two or three weeks, if the person survives, the whole of it may be removed from the body, with the exception of traces in the hair, nails and bones. In 1891 Sir T. Stevenson found it in the urine for four days only after the taking of a poisonous dose of arsenic, and consequently the amount found on analysis need not by any means correspond in any degree with the amount which was administered.

It is apparently true that arsenic is not a natural constituent of the body at birth. Nevertheless there are so many unsuspected sources of arsenic, and means by which it may enter the body in minute quantities (and analysis can detect a minute fraction of a grain), that caution is necessary in swearing in a given case that arsenic was the cause of death. So strongly has this been felt, that in 1904 a man was set free in France who had been condemned some years previously on evidence of this sort to a long term of imprisonment. The case is thus reported in the *Daily Telegraph*, February 29th, 1904 :—

The Judicial Committee of Revision of Trials has allowed the chemist Danval, who received a free pardon eighteen months ago, to bring on a new trial to clear himself completely. He was found guilty twenty-five years ago of the murder of

[1] This statement requires modification. If repeated doses are given at intervals insufficient for the complete elimination of each dose, arsenic must accumulate in the body ; with longer intervals it would not.

his wife by having poisoned her with arsenic, and was sentenced to transportation for life. The evidence on which Danval was found guilty was purely scientific, and consisted in the fact that 1 milligram (0·015 of a grain) was found in her body.

Bamford,[1] in Egypt, regarded a concentration of up to 0·5 mg. per 100 g. tissue (5 parts per million) as "normal" for viscera, and van der Rieb Copeman and Kamerman[2] state that (in South Africa) hair normally contains about that amount.

We may sum this aspect of the matter up briefly by saying that to prove death from arsenic poisoning some symptoms known to be caused by arsenic must have been observed before death, and leading up to death, and there must be unequivocal proof that some reasonable quantity, *i.e.*, at least a grain or a large fraction of a grain of the metal, was found in the viscera, or good evidence of such a lapse of time after the administration of the last dose as to give a satisfactory explanation of its possible absence. Finally, it must be remembered that a person may die of a disease for which arsenic is quite legitimately being administered. In Taylor's time it was a sort of mystery that large quantities of arsenic could be administered homicidally. We now know that such is quite possible, in gruel or thick liquid or even in cake or bread, for arsenic in some forms is practically tasteless; suicides have been known to swallow as much as 40 ounces.[3]

Sir T. Stevenson has known an ounce of arsenic homicidally put into a pint of rice pudding. The pudding was eaten without suspicion.[4]

Taylor thought it worth his while to lay stress on the fact that arsenic had been found in bodies that had been buried for eight and even fourteen years, but considering that arsenic is a metallic element there seems to us nothing wonderful in such a fact; on the contrary, it would seem more miraculous if no arsenic were found even after a thousand years if the remains had not been disturbed.

The condition of the arsenic found in the stomach should, if possible, be noticed. A witness should be prepared to say whether it is in fine powder or coarse fragments; whether it is mixed with soot or indigo, or whether it is in the ordinary state of white arsenic. These points may be material as evidence in reference to proof of possession, of purchase, or administration.

Chemical Analysis (*Arsenic as a solid*). In the simple state, *white arsenic* may be identified by the following properties: 1. White arsenic, when heated to a moderate temperature (about 400° C.), is vapourised without first passing through the liquid state. Commercial samples may not disappear completely when heated in this way, since they frequently contain non-volatile impurities, such as plaster of paris, chalk, etc. If the heating is carried out slowly in a narrow-bore glass tube, the arsenious oxide vapour condenses on the cooler parts of the tube in the form of minute octahedral or tetrahedral crystals, remarkable for their lustre and brilliancy. The shape and appearance of the crystals differentiates

[1] "Poisons," London 1938.
[2] *Loc. cit.*
[3] *Pharm. Jour.*, 1872, p. 75.
[4] *R.* v. *Leffley*, Lincoln Ass., November 1884.

sublimed arsenious oxide from sublimates of other substances—corrosive sublimate, calomel, oxalic acid, antimony oxide—which are obtained at a similar temperature. Under a microscope magnifying 250 diameters, the appearance of these crystals is remarkably beautiful and characteristic; one not exceeding the four-thousandth of an inch in diameter may be easily recognised by the aid of this instrument. They may be measured even to the sixteenth-thousandth of an inch in diameter. It will be observed in these experiments that the vapour of white arsenic is odourless. 2. On boiling a small quantity of the powder in distilled water, it is not readily dissolved, but it partly floats in a sort of white scum, while a part becomes aggregated in lumps at the bottom of the vessel. It requires long boiling in order that it may be dissolved and equally diffused through water. This property of arsenic has given rise to some important questions at criminal trials. The floating of arsenic takes place whether the water is hot or cold, and whether the water is added to the poison or the poison to the water.[1] This property has attracted attention, and in one instance was the means of saving life. 3. When the powder is treated with a weak solution of sulphide of ammonium in a watch-glass, there is no change of colour, as there is with most metallic poisons; on warming the mixture the white powder is dissolved; and on continuing the heat until the ammonium salt is expelled a rich yellow or orange-red film is left (arsenious sulphide), which is soluble in alkalies, and insoluble in hydrochloric acid. If, however, a solution containing hydrogen sulphide and hydrochloric acid be used instead of ammonium sulphide, a yellow precipitate of arsenious sulphide appears at once, or on gently warming the mixture. 4. Heated on platinum wire in a smokeless flame, the powder imparts to it a pale blue colour, while it is volatilised in white fumes. 5. Another test is stannous chloride in hydrochloric acid. The mixture is brought to the boiling point, and it should remain colourless. If the hydrochloric acid contains a trace of arsenic, the liquid will acquire a light brown colour. On adding a minute quantity of solid arsenious acid, this is dissolved, and metallic arsenic is deposited in the form of a brown or brownish-black precipitate. A salt of antimony is not thus affected. 6. **Reduction test.** Any solid compound of arsenic (*e.g.*, arsenites, including the green arsenical pigments; the sulphides; the chlorides; and the arsenates as well as white arsenic) is reduced to metallic arsenic when heated with a mixture of sodium carbonate and potassium cyanide or carbon. A mixture of three parts of anhydrous sodium carbonate with one part of potassium cyanide gives very good results; an almost equally good flux may be prepared by incinerating sodium acetate or sodium tartrate in a closed vessel; in case of necessity finely powdered potassium ferrocyanide may be used. The arsenic-containing substance is introduced into a test-tube, which, about half an inch from the bottom, has been drawn out into a narrow tube of about one-tenth inch bore. The arsenical powder is then covered with about six times its bulk of flux, and the tube, with its contents, is gently heated to expel moisture. Strong heat is then applied first to the flux and finally to the arsenic. The metallic arsenic which is produced sublimes on the narrow part of the tube, forming a black deposit which shades off into brown or grey. Antimony compounds give no deposit of the metal when treated in this way, but compounds of mercury, cadmium, tellurium and selenium do.

[1] See the case of *R.* v. *Smith*, Wells Lent Ass., 1869.

Mercury, of course, may be at once distinguished by examining the deposit under the microscope—the mercury deposit consists of or is bordered by definite globules. The other metallic deposits, like that of arsenic, are crystalline. Arsenic, however, may be completely distinguished by the following tests :—

(*a*) Break the tube below the deposit. Heat the deposit gently. It is possible, by careful regulation of the heat, to drive away the brown deposit first, leaving only the black or grey mirror of metallic arsenic. On further heating this also disappears, and in both cases white arsenious oxide is deposited on the upper, cool part of the tube. This, on microscopic examination, is seen to consist of characteristic lustrous octahedra and tetrahedra. This test is absolutely characteristic of arsenic, and distinguishes it from all other sublimates, whether of metals or non-metals.

(*b*) The metallic sublimate, or the crystals produced from it, may be subjected to the following process :—Break the glass on which the sublimate is deposited into fragments, and digest these in a few drops of fuming nitric acid, previously proved to be free from arsenic. The sublimate is thereby converted into *arsenic acid*. The acid solution should be evaporated to dryness : the white non-crystalline residue obtained should be dissolved in a few drops of distilled water, and a strong solution of nitrate of silver, or of the ammonio-nitrate, added in small quantity to the residue. A brick-red coloration indicates arsenic acid, and thus proves incontestably that the sublimate was of an arsenical nature.

The *process of reduction*, with the corroborative results above mentioned is, when thus applied, conclusive of the arsenical nature of the substance under examination.

Arsenic in solution in water. Liquid tests. The solution of arsenious acid is clear, colourless, possesses scarcely any perceptible taste, and has but a feebly acid reaction. In this state, we should first evaporate slowly a few drops on a glass slide, when a crystalline residue will be obtained. On examining this with a microscope, it will be found to consist of numerous minute octahedral crystals, presenting triangular surfaces by reflected light.

Of the following tests, the first three are of value when relatively large amounts of arsenic are being dealt with. The fourth, Reinsch's test, is much more delicate, while the fifth and sixth are capable of detecting even mere traces of arsenic, and are applicable to the quantitative estimation of minute amounts such as may be met with in medico-legal work.

1. **Silver Test.** On adding to the solution *an ammoniacal solution of silver nitrate*, a pale yellow precipitate of arsenite of silver falls, changing, under exposure to daylight, to a greenish-yellow colour. The test is made by adding to a strong solution of nitrate of silver a weak solution of ammonia, and continuing to add the latter until the brown oxide of silver, at first thrown down, is almost redissolved. The yellow precipitate is soluble in nitric, tartaric, citric, and acetic acids, as well as in excess of ammonia. It is not dissolved by potash or by soda.

2. **Copper Test.** On adding to a solution of arsenic *ammonia-sulphate of copper*, a light green precipitate of arsenite of copper is formed, the tint of which varies according to the proportion of arsenic present,

and the amount of reagent added : hence if the arsenic is in small proportion, no green precipitate at first appears ; the liquid simply acquires a blue colour from the reagent. Within an hour, if arsenic is present, a bright green deposit is formed, which may be easily separated from the blue liquid by decantation. This test is made by adding ammonia to a weak solution of sulphate of copper until the bluish-white precipitate, at first produced, is nearly redissolved : it should not be used in large quantity if concentrated, as the deep blue colour tends to obscure or conceal the green precipitate formed. The precipitated arsenite of copper is soluble in all acids, mineral and vegetable, and in ammonia, but not in potash or soda. If a small quantity of the blue ammoniacal solution of this precipitate is poured over a crystal of nitrate of silver, a film of yellow arsenite of silver will appear around the crystal. If a strong solution of silver nitrate is added to the blue liquid, nearly neutralised by diluted sulphuric acid, a yellow precipitate of arsenite of silver is also produced. Thus the silver and copper tests may be employed with the same specimen of liquid. The *dried* precipitate of arsenite of copper, when slowly and moderately heated in a well-dried reduction-tube, yields a ring of octahedral crystals of arsenious acid—black cupric oxide being left as a residue.

3. **Sulphuretted Hydrogen Test.** Sulphide of ammonia gives no precipitate in a solution of arsenic until an acid has been added, by which property arsenic is distinguished from most metallic poisons. On adding dilute hydrochloric acid, a bright lemon-yellow coloured precipitate is thrown down (orpiment or arsenious sulphide). It is better, however, to employ, in medico-legal analysis, a current of washed sulphuretted hydrogen gas, which is easily procured by adding to ferrous sulphide, or, better, barium sulphide, which can readily be obtained free from arsenic, in a proper apparatus, a mixture of one part by volume of strong hydrochloric acid and one part of water. The arsenical liquid should be slightly acidified with pure diluted hydrochloric acid, *before* the gas is passed into it : and care should be taken that it is not alkaline. The yellow compound is immediately produced if arsenic is present, and it may be collected after boiling the liquid sufficiently to drive off any surplus gas. The precipitation is likewise facilitated by adding to the liquid a solution of chloride of ammonium. The yellow precipitate is known to be arsenious sulphide by the following properties :—1. It is insoluble in water, alcohol, and ether, as well as in dilute hydrochloric acid, and vegetable acids : but it is decomposed by strong nitric and nitro-hydrochloric acids. 2. It is dissolved by potash, or ammonia : forming, if no organic matter is present, a colourless solution. 3. When dried and heated with two or three parts of a mixture of carbonate of sodium and cyanide of potassium, it gives a sublimate of metallic arsenic. Unless these properties are possessed by the yellow precipitate formed by sulphuretted hydrogen in an unknown liquid, it cannot be a compound of arsenic. On the other hand, when these properties are possessed by the precipitate, it must be arsenic, and can be no other substance.

4. **Reinsch's Test.** In the application of this test, the liquid suspected to contain arsenic, or the solid dissolved in distilled water, is boiled with about one third of its volume of *pure* hydrochloric acid (free from arsenic), and a small slip of copper is then introduced. The copper must be first

proved to be free from arsenic, as this is a very common contamination. Pure electrolytic copper, free from arsenic, must be used in every case where it is available. If arsenic is present in the liquid, even in small quantity, the polished copper acquires either immediately, or within a few minutes, an iron-grey metallic coating from the deposit of this metal. This is apt to scale off if the arsenic is in large quantity, or if the liquid is very acid, or long boiled. Remove the coated slip of copper, wash it successively in water, alcohol and ether, dry and gently heat it in a reduction-tube, when arsenious acid will be sublimed in minute octahedral crystals: if these should not be apparent from one piece of copper, several may be successively introduced. When the quantity of arsenic is very small, the polished copper merely acquires a faint grey or bluish tint. The deposit is in all cases materially affected by the degree of dilution, and sometimes it will appear only after the liquid has been much concentrated by evaporation. Excessive evaporation, however, must be avoided, for at hydrochloric acid concentrations greater than 12 per cent. (*i.e.*, about one part of concentrated acid in three of the diluted mixture) there is a danger of losing arsenic by volatilisation. We are not obliged to dilute the liquid in the experiment and there is no material loss of arsenic, as in Marsh's process; the whole may be removed and collected by the introduction of successive portions of pure copper. This process is extremely delicate, and the results are speedily obtained. Among the cautions to be observed are these: – (1) not to employ too large a surface of copper in the first instance; and (2) not to remove the copper from the liquid too soon. When the arsenic is in minute quantity, and the liquid is much diluted, or not sufficiently acidified, the deposit sometimes does not appear for half an hour. If the copper is kept in for an hour or longer, it may acquire a dingy tarnish from the action of the acid and air. It is essential to identify the deposit as arsenic by converting it to arsenious oxide as described above. When examined by a quarter-inch power under the microscope, the octahedral crystals may be seen and recognised by their shape. The smaller the crystal the more perfect the form. If the copper with the deposit, and the tube, have not been well dried, or if the upper part of the tube is too cold so that deposition of the crystals occurs too soon, the angularity of form is not distinct. The test cannot be considered positive unless these crystals are obtained. The crystals may be tested by the processes already described.

The mere fact that a grey deposit is formed upon pure copper when boiled in the liquid under examination after acidification with pure hydrochloric acid, affords no absolute proof of the presence of arsenic. Other metals, *e.g.*, antimony, mercury, silver and bismuth, all yield deposits with Reinsch's test; and the grey deposit yielded by bismuth may readily be mistaken for that of arsenic. The volatility of the arsenical deposit, the crystalline nature of the sublimate, and its reaction when treated successively with nitric acid and nitrate of silver, must in all cases be ascertained before it is concluded that the deposit is arsenical.

In consequence of the errors into which faulty methods of employing Reinsch's test have led, its reliability has been much discredited: and though in skilful hands the results obtained by it are trustworthy, it would perhaps be unsafe to rely upon it in an important criminal

investigation. It may be conceded that Marsh's process will detect a smaller quantity of arsenic than the process of Reinsch ; but the latter, when the quantity of liquid is small, and pure materials are used, will detect the one-hundred-and-fiftieth or the two-hundredth part of a grain of the poison. It appeared to the author that in a criminal case it would not be safe to depose to the presence of arsenic from Marsh's process alone, when the quantity of poison was *too small* to admit of separation or corroboration by the process of Reinsch. Conversely the results of Reinsch's should be corroborated by those of Marsh's process. It was this over-reliance on the extreme delicacy of Marsh's process in researches where it admitted of no corroboration whatever that led Orfila to assert that arsenic was a natural constituent of the human body.[1]

Arsenates give a positive result in the Reinsch test only very slowly or when in moderately high concentration. The test may therefore fail if oxidising agents—*e.g.*, chlorates, manganese dioxide—are present. Many organic compounds of arsenic also give little or no deposit of arsenic on the copper foil.

The following is a simple method of detecting arsenic in copper. Add to pure hydrochloric acid, diluted with six parts of water, one or two drops of a weak solution of ferric sulphate or chloride. Boil the acid liquid and introduce the copper, well cleaned and polished, into the boiling liquid. Arsenical copper acquires a dark tarnish, while non-arsenical copper retains its red colour. It will be found from this experiment that copper in the state of gauze or fine wire generally contains arsenic. This would present no obstacle to the detecting of arsenic by it, provided the copper gauze were not dissolved. Arsenic can only be separated from its copper alloy by the destruction of the alloy and the solution of the two metals.

5. **The Marsh-Berzelius Test.** The most useful method of determining small quantities of arsenic is the Berzelius modification of the Marsh test.

When arsenic, in the arsenious condition, is introduced into a hydrogen generator, arseniuretted hydrogen is liberated and may be burned in a jet together with the escaping hydrogen. If the flame is suddenly cooled as, *e.g.*, by placing a piece of cold porcelain in the flame, arsenic is deposited as a brown stain, which is soluble in a solution of hypochlorite (bleaching powder). The black stain obtained in the same conditions from solutions containing antimony is insoluble in this reagent. This is the original Marsh test. Berzelius suggested the heating of a part of the exit tube so as to cause the decomposition of the arseniuretted hydrogen and the deposition of elementary arsenic on the cooler part of the tube. Antimony is deposited under the same conditions as arsenic, but is, as mentioned above, insoluble in bleaching powder solution. Moreover, the arsenic stain is brownish at the edges and is deposited at a little distance from the flame, whereas that of antimony has a tin-like lustre and appears close to (sometimes on both sides of) the heated part of the tube. The apparatus should consist of a conical flask fitted with a tap funnel and an exit tube, preferably fixed to a ground glass stopper. The exit tube is connected to a drying tube filled with arsenic-free calcium

[1] *Vide* also the Report of the Arsenic Commission, p. 431.

chloride between plugs of glass wool, and the further end of the drying tube is joined to a horizontal exit tube of transparent silica which terminates in a turned up jet.

Hydrogen may be liberated by the action of pure dilute sulphuric acid on pure zinc activated by the addition of cadmium chloride. The generating flask should be cooled in ice to prevent the evolution of gas

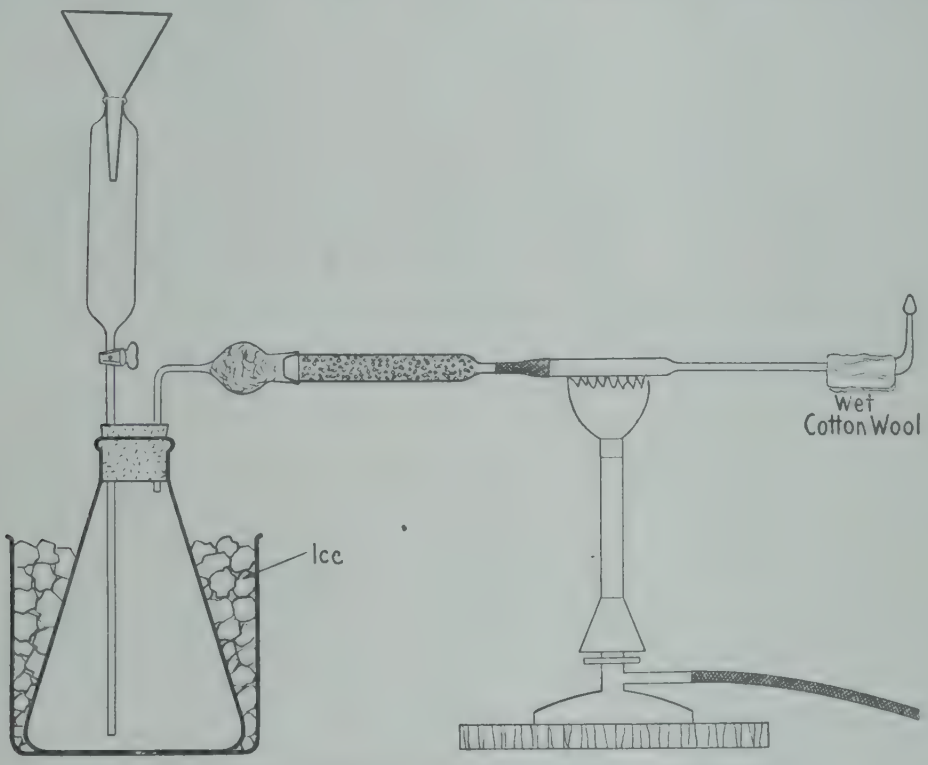

Fig. 1. Marsh-Berzelius test for arsenic; zinc and acid method.

from becoming too rapid. Before commencing the operation the joints must be examined to ensure that they are gas-tight, and the evolution of hydrogen should be allowed to continue for about fifteen minutes to drive all air out of the apparatus. The hydrogen may then be safely burned at the end of the exit tube. During this time part of the exit tube should be heated to dull redness, while the narrow part near the turned up tip is kept cool by being wrapped in wet filter paper. After 15–20 minutes, no trace of dimness must be visible in the cool part of the tube examined against a white background. Then the suspected solution, which has been previously boiled with stannous chloride to reduce any arsenic present to the arsenious condition, is added. Nitrates and nitrites must be absent; silver and mercury salts also interfere.

The volume of the liquid to be tested should be measured and a small aliquot fraction should be added first. If necessary to prevent frothing, a few drops of pure alcohol may also be added. No more of the solution should be used than is necessary to produce a mirror of arsenic comparable in size with standard mirrors previously prepared. A convenient series of arsenic mirrors must be available. These should be prepared in standard tubes from solutions containing from 0·00002 grm. to 0·0005 grm. of arsenious oxide. When a mirror of a suitable size has been produced, or when the whole of the suspected solution has been added, the tap funnel should be washed with dilute acid and the process continued

for a further ten or fifteen minutes. The comparison of the mirror with the standards may then be made, and the quantity of arsenic reported as the number of milligrams of arsenious oxide present in the whole of the material under examination.

The quantitative estimation is never very accurate owing to the difficulty of making strictly comparable "mirrors" and of detecting very small differences by visual examination. With relatively large amounts (of the order of 5 mg.) it is possible to weigh the deposit on a micro-balance, and so achieve greater accuracy.

The Electrolytic Modification of the Marsh Test. This modification was suggested as a means of ensuring the generation of arsenic-free hydrogen. The apparatus devised by Thorpe[1] consists of an electrolytic cell with a perforated cone of thin lead-foil as cathode, and a strip of platinum-foil as anode. The cathode is suspended in the cathode-compartment by means of a platinum wire fused through the glass stopper. The platinum-foil of the anode is wrapped around the porous pot which forms the lower part of the cathode-compartment. The anode-

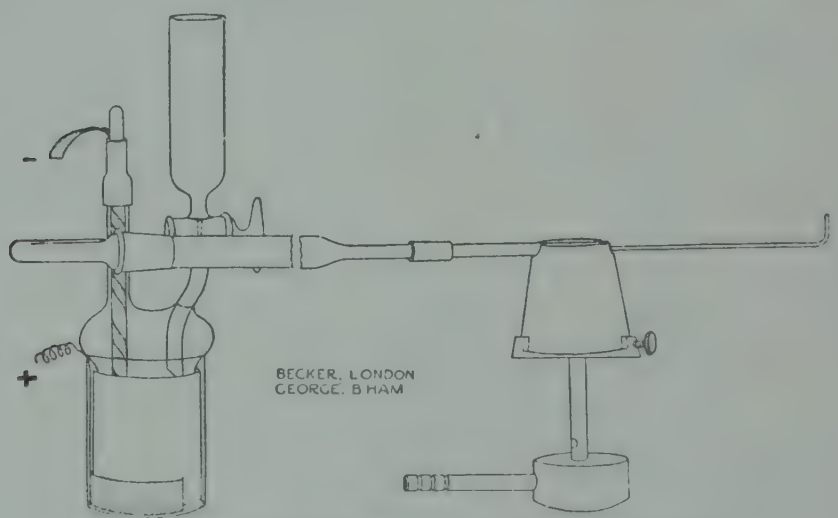

Fig. 2. Marsh-Berzelius test for arsenic; electrolytic method.

compartment consists of an outer glass vessel. Both compartments contain pure, dilute sulphuric acid, and the level of the liquid should be about 2 or 3 centimetres from the bottom in the inner vessel, and about 0·5 cm. higher in the outer vessel. A current of 2 to 3 amperes at a potential of 7 volts should be passed for about twenty minutes.

On the test for arsenic the *Lancet* thus comments on the report of the Manchester Commission :—

"The Commission has received a great deal of chemical evidence, especially from two strong committees, one a joint committee of the Societies of Public Analysts and Chemical Industry, with Mr. Otto Hehner as chairman, and the other, with Dr. Thorpe as chairman, appointed by the Board of Inland Revenue to advise as to arsenic in beer ingredients. The report deals with the essential principles of applying what is now termed the 'Marsh Berzelius test' to various substances. In this test, as is well known, the arsenic from the arseniuretted hydrogen is deposited, not on a porcelain tile, as in the 'Marsh' test, but in a capillary tube.

[1] *Proc. Chem. Soc.*, 1903, 19, 183.

Within a certain range 'mirrors' of arsenic deposited in these tubes show definite differences in intensity according to the quantity of arsenic present, and an estimation is made by comparing the intensity of the mirror obtained from a given weight of substance with a set of standard mirrors. Attention to various points of detail is essential to accuracy. In nearly all cases it is necessary in the first instance to destroy completely any organic matter present. The presence of iron salts in the Marsh apparatus may seriously vitiate the result. It is not only necessary to have zinc which is free from arsenic, but also zinc which is 'sensitive' and does not retain the arsenic in the solution tested. The Commission finds that chemists are now agreed as to these and other sources of error which is necessary to avoid, and that if due regard is had to these points differences as small as 0·2 part of arsenic per million in the substance taken or 0·0014 grain of arsenic per pound can be readily distinguished. . . . The presence of arsenic will be detected when in amounts well below $\frac{1}{1000}$ of a grain per pound or $\frac{1}{7000}$ of a grain per gallon. The departmental committee has found that in the case of beer ingredients many of the difficulties attending the Marsh-Berzelius test are obviated by an electrolytic method of evolving arseniuretted hydrogen, which can be applied wherever a current of sufficient intensity is available."

6. **The Gutzeit Test.** Although this test is not characteristic of arsenic, it permits very small quantities of arsenic to be detected in the absence of antimony, hydrogen sulphide, sulphur dioxide or phosphine.

Arseniuretted hydrogen is produced in a hydrogen generator, as in the Marsh test, iodine being added until the solution is yellow, to remove sulphur dioxide and hydrogen sulphide. The gas is dried by calcium chloride and allowed to pass through a glass tube of about 4 mm. bore containing a strip of paper which has been moistened with solution of silver nitrate and dried.

The paper is turned yellow in the presence of arsenic, owing to the formation of a double compound of silver nitrate and silver arsenide. The addition of ammoniacal solution to the paper causes the separation of metallic silver, the stain being turned black.

The method yields excellent quantitative results if standard stains are prepared for comparison on silver nitrate paper, using known quantities of arsenious oxide dissolved in dilute hydrochloric acid. These standard stains are not permanent, and should be prepared as required.

A modification of the test which increases its sensitivity is to use mercuric chloride paper instead of silver nitrate paper. The (filter) paper is soaked in a 5 per cent. solution of pure mercuric chloride, dried, cut into strips about 7 cm. long and 4 mm. wide, and stored in the dark in tightly stoppered glass bottles containing a little calcium chloride.

A distinct yellow stain is given by ·003 mg. of arsenic. The colour is deepened by immersion in hydrochloric acid for two minutes at a temperature not above 60° C., and the stain turns black when the paper is soaked for a few minutes in ammonium hydroxide. Antimony gives a grey or black stain at once (never yellow).

Evers[1] has described an electrolytic modification of the Gutzeit test, using leaden electrodes, and as electrolyte dilute sulphuric acid (15 to

[1] *Chemist and Druggist*, 1926, *105*, 259.

20 per cent. by weight) containing 0.025 per cent. of cadmium sulphate. A direct current of 3 to 6 amperes with an E.M.F. of 7 to 9 volts is used.

This method is specially recommended for the examination of drugs, many of which may be tested directly for arsenic without preliminary treatment.

7. **Iodometric Determination of Arsenic.** Several methods have been evolved in recent years for the titrimetric determination of arsenic. They depend on the separation of arsenic as arsine, the absorption of this in silver nitrate solution, and titration of the arsenite so formed with iodine. The technique described by Levvy[2] permits the determination, in biological material of 5–50 μg. (·005 to ·05 mg.) arsenic, with, in a single determination of 20 μg. a standard deviation of 3·7 per cent. This is better than is claimed by many authors for stain-matching methods and titrimetry is much more objective. The sensitivity is of the same order as that of the Marsh and Gutzeit methods.

For a detailed discussion of the method, the original paper by Levvy should be consulted. The procedure, he describes as follows :—

" *Digestion—small scale.* Up to 5 ml. blood, 10 ml. urine or 2 g. tissue are measured into a 100 ml. Kjeldahl flask, and conc. HNO_3 (5 ml.), $HClO_4$ (3 ml., 60 per cent. *w/v*), conc. H_2SO_4 (10 ml.) and two or three clean glass beads are added. The flask is warmed, with shaking, over a naked flame till brown fumes are given off, and heating is continued on a digestion stand over a very small flame (to avoid bumping) till they are no longer evolved. The contents are then gently boiled till H_2SO_4 fumes appear, and the clear liquid becomes colourless. Heating is continued for 10 min. If the mixture chars at any time, the flask is cooled slightly and a few drops of fuming HNO_4 added.

When digestion is complete, the flask is allowed to cool and saturated $(NH_4)_2C_2O_4$ solution (5 ml.) is added. The liquid is brought to the boil and heating continued till white fumes appear. Distilled water (5 ml.) is added to the cooled liquid, which is again brought to the boil, and heating continued till 5 min. after the first appearance of white fumes.

Control experiments, with reagents alone, are carried out in exactly the same manner. It is important that heating of the digest at each stage should be continued for at least the period specified.

Digestion—large scale. Tissue. Up to 20 g. of minced, moist tissue are wrapped in very thin paper and placed in a 300 ml. Kjeldahl flask (with ground joint). After the addition of conc. HNO_2 (30 ml.) and two or three clean glass beads, the flask is set aside overnight.

Blood. Up to 30 ml. blood are treated as described above.

Urine. To not more than 150 ml. urine one-fifth its volume of fuming HNO_3 and two or three glass beads are added, the liquid evaporated to about 10 ml., and the digestion then carried out.

$HClO_4$ (15 ml., 60 per cent. *w/v*) and conc. H_2SO_4 (20 ml.) are added to the flask, which is warmed cautiously with shaking over a naked flame till reaction sets in, and is then placed on a digestion stand. After the first violent reaction subsides, the burner is lit and the contents brought to the boil. When digestion is complete, 20 ml. of saturated $(NH_4)_2C_2O_4$ solution and of water are added. It may be necessary to make several additions of fuming HNO_3 before digestion is complete.

[2] Levvy, G. A. (1943), *Biochem. J.*, *37*, 598.

$AsCl_3$ distillation. This is necessary only after large-scale digestion. A delivery tube is connected, with conc. H_2SO_4 as lubricant for the ground-glass joint, to a 150 ml. Fresenius flask containing distilled water (40 ml.) immersed in ice-water (*see* Bang, 1925).

Distilled water (50 ml.) is added to the Kjeldahl flask and the contents are cooled thoroughly. Through a wide-bore funnel, $FeSO_4$, $7H_2O$ (8 g.), KBr. (2 g.) and NaCl (25 g.) are added and the flask immediately connected to the previously lubricated joint of the delivery tube. A 200 ml. volumetric flask is placed over the exit of the Fresenius flask. The liquid in the Kjeldahl flask is boiled vigorously for exactly 10 min., the ice around the receiver being renewed as necessary. With the liquid still boiling, the delivery tube is disconnected from the Kjeldahl flask. The contents of the Fresenius flask are then transferred to the 200 ml. volumetric flask, and, when cool, diluted to the mark.

Separation of As as AsH_3. Two absorption tubes, the second of which acts as a guard tube, are charged with N/50 $AgNO_3$ (1·5 ml.). The tubes are stoppered and connected to a small wash-tube containing 10N NaOH (renewed after 12 estimations), glycerine being used as a lubricant for rubber tubing. A right-angle tube, bearing a stopper to fit the AsH_3 generation flask, is attached to the wash-tube.

After a small-scale digestion, the contents of the Kjeldahl flask are transferred to a 150 ml. conical flask, with 40 ml. distilled water as washing fluid. Alternatively, a measured fraction of the diluted digest is made up to 50 ml. with 20 per cent. H_2SO_4. 40 per cent. $SnCl_2$ (10 drops) and 5 per cent. $CuSO_4$ (3 drops) are added. The liquid is warmed if necessary, Zn shot (15 g. freed from dust and of fairly uniform size) quickly introduced, and the flask at once connected to the previously prepared absorption apparatus.

After a large-scale digestion, if the portion taken is less than 100 ml., it is made up to this volume in a 150 ml. conical flask with 20 per cent. H_2SO_4. When the total distillate is used, it is transferred to a 250 ml. conical flask. 40 per cent. $SnCl_2$ (10 drops/100 ml. Total vol.) and 5 per cent. $CuSO_4$ (3 drop/100 ml. total vol.) are added and the liquid is warmed. Zn (25 g.) is introduced and the flask immediately connected to the absorption apparatus.

Titration of arsenite. When H_2 has been passed through the absorption apparatus for 30 min. the tubes are disconnected. The rubber stopper of the absorption tube is washed down with a few drops of distilled water, and the gas inlet is connected to the compressed air. KI is added till the precipitated AgI redissolves. After the addition of a spatula-point of $NaHCO_3$ and a drop of starch solution the tube is slipped over the jet of the Conway burette. It is held in place by sliding the titration platform under it, and with air passing through the liquid at a gentle rate I_2 is run in till a red-brown colour appears and persists for 30 sec. Before the end of the titration, the gas exit is washed down by increasing the flow of air till a little of the liquid is blown into it. A daylight lamp is of advantage in observing the end-point. If the liquid in the guard tube darkens, it is titrated in the same way.

At the beginning of each day, the glass tubing in the burette is thoroughly washed out with fresh I_2 from the reservoir. To standardise the I_2, two different quantities of arsenite solution are measured into

absorption tubes, the volume made up to about 2 ml. with N/50 $AgNO_3$, and the titrations carried out as described above. The amount of As (μg.) is plotted against ml. I_2, and the analytical results are read off from the straight line joining the two points. This line cuts the I_2 axis at the volume required to cause a colour change in 2 ml. N/50 $AgNO_3$ treated as above, in absence of arsenite."

Arsenic in Solids or Liquids containing Organic Matter

The tests which must be applied are those already detailed. This section is concerned with the methods which must be used, as a preliminary, to obtain material suitable for analysis, when organic matter is present, as in the examination of viscera, stomach contents, etc.

The arsenic may be mixed with the organic liquid in the form of heavy lumps or powder. The great specific gravity of this substance allows of the liquid being poured off, and the sediment collected. When washed and dried, it may be found to be crystalline. It should be weighed, and then tested by the processes elsewhere described. Let us assume that the organic liquid is milk or beer ; it will be necessary to determine whether any arsenic is dissolved in it. Filter a portion ; place it in a dialysing tube and immerse the mouth of the tube in distilled water. In a few hours the arsenic will have traversed the membrane, and will be found in a clear and nearly colourless solution in the water. Tests may be then applied to this liquid for the detection of arsenic. They should never be applied directly to coloured organic liquids.

In testing *solids* generally for arsenic, we may employ the process of Reinsch as a preliminary test. The solid is boiled in water acidified with one-third of its volume of pure hydrochloric acid, until it is either dissolved or its structure broken up. A small portion of pure polished copper is then introduced. In a few minutes, if arsenic is present—even to the extent of the thousandth part of a grain—there will be a metallic deposit on the copper, and this will yield crystals when heated in a tube. *Liquids* suspected to contain arsenic may be treated in a similar manner. Water is not required ; the liquid is simply acidified with one-sixth part of pure, strong hydrochloric acid. If the solution of the organic solid or the organic liquid is not deeply coloured, the stannous chloride test may be employed in place of the process of Reinsch. Less than the sixtieth part of a grain, even under considerable dilution, may thus be readily detected. The test reveals traces of arsenic in ordinary sulphuric and hydrochloric acids.

Precipitation as sulphide. When arsenic has been introduced into an organic liquid in large quantity, it may be directly precipitated as sulphide by a current of washed sulphuretted hydrogen. In case arsenates are present, it is advisable to add a little potassium iodide to the liquid before passing in the sulphuretted hydrogen. This greatly aids precipitation of arsenic sulphide in these circumstances. The liquid should be boiled, filtered, and acidified with pure hydrochloric acid before the gas is passed into it. When precipitation has ceased, it should be again filtered, the precipitate collected, washed successively with water, alcohol, and ether, dried and weighed. By operating on a measured portion of the solution, the amount of white arsenic may be determined approximately by the weight of the yellow sulphide obtained—100 parts by weight of sulphide being equal to 80·49 parts of white arsenic. The properties of the yellow

precipitate should be verified according to the methods mentioned above. In some cases arsenic may be present, but in a quantity too small to be precipitated as sulphide by sulphuretted hydrogen. In others the presence of other metals of the same group—lead, mercury, etc.—may interfere with or prevent precipitation. The presence of any free alkali in a liquid prevents the formation of a precipitate—hence the injunction to acidify.

This method of estimation, even when the amounts of arsenic permit its use, is best made after destruction of organic matter by one of the following processes, and only if interfering metals are absent.

When white arsenic is found, in powder, as a sediment in organic liquids, it is obvious that it must have been taken in the solid state, and, although mixed with a liquid or solid, still in an undissolved form. If found only dissolved, it may have been taken either in solution or in a solid form—the dissolved portion being part of the solid taken up by the fluids of the stomach, and the remainder having been expelled by vomiting and purging. This question was of importance in *R.* v. *Sturt*.[1] The deceased, in this case, died from the effects of arsenic administered in the form of a powder and, it was believed, in a mince-pie. It was suggested that the poison might have been swallowed in ginger-beer, but then it could not have been in solution: it must have been mechanically mixed with the liquid. The judge who tried this case was apparently not aware of any difference existing between the actual solution and the mechanical suspension of a solid in a liquid.

Distillation process.—When the poison is in so small a quantity that it does not admit of precipitation by sulphuretted hydrogen, and no solid particles of arsenic are found in the stomach, in its contents, or in any article of food, another method may be resorted to for detecting its presence. This method equally applies to the detection of arsenic deposited as a result of absorption in the soft organs of the body, as in the liver, kidney, or heart, and to arsenic in all forms, except in pure insoluble sulphide or orpiment. Although, after long interment, white arsenic passes, more or less rapidly, into the state of yellow sulphide as a result of chemical changes during putrefaction, the conversion is generally only partial. The only condition for success is, that the substance, whether food, blood, mucus, the liver, or other organ, should be first thoroughly dried, either by exposure to a current of air, or in a water-oven. The dried solid should then be powdered in a mortar and placed in a flask of sufficient capacity, with enough of the strongest fuming hydrochloric acid to cover it completely. [The freedom of this acid from arsenic should be first carefully determined.] The complete separation of arsenic from organic substances depends greatly on their perfect desiccation, and on the concentration of the acid employed. After twenty-four hours' digestion at room temperature the retort or flask containing the mixture—which should be of such a size that the materials should not fill it to more than one-third or one-half of its capacity—should be fitted with a water-cooled condenser, and then gradually heated on a sand-bath until the acid liquid begins to pass over. A small flask cooled in ice, and provided with a loosely fitting cork may be employed to collect the product. This should contain a small quantity

[1] Lewes Lent Ass., 1863.

of distilled water into which the end of the condenser tube dips, so as to fix and condense any vapours that may pass over. The distillation may be carried to dryness, or nearly so, on a sand-bath ; and it is advisable, in order to ensure the separation of the whole of the arsenic as chloride, to add to the residue in the retort another portion of pure concentrated hydrochloric acid, and again distil to dryness. The author found, however, that portions of dried liver and stomach gave up every trace of arsenic by one distillation when a sufficient quantity of hydrochloric acid had been used, and the process slowly conducted by a regulated sand-bath heat.

The liquid product may be coloured, turbid, and highly offensive if distilled from decomposed animal matter. Exposure to the air for a few hours sometimes removes the offensiveness, and there is a precipitation of sulphur, or of some sulphide, which may cause a partial loss of arsenic. The distillate may be separated from any deposit by filtration, and, if still turbid, it may be again distilled to separate it from any organic matter that may have come over.

If arsenic was present in the solid, the distillate will be a solution of arsenious acid in hydrochloric acid. The quantity of dry organic substance used in the experiment must depend on the quantity of arsenic present, as revealed by a preliminary trial with Reinsch's process. If large, two or three drachms of the dried substance, or even less, will yield sufficient arsenic for further proceedings. For the absorbed and deposited poison, half an ounce of the dried organ, corresponding to two ounces of the soft organ, will frequently suffice ; but a negative conclusion of the absence of arsenic should not be drawn from a smaller quantity than two to four ounces of the dried substance, whether liver, kidney, or heart. If oily matter should be distilled over, this may be separated by passing the distillate through a paper filter wetted with water.

The author found this process efficient for procuring a clear solution of chloride of arsenic from such different substances as ordinary food, the liver and other soft organs, the scalp of the head, blood, contents of the stomach, arsenical wall-papers, metallic copper, blue vitriol, and various mineral powders. He thus discovered arsenic in two ounces of the earth of cemeteries, as well as in the mud of rivers, in spite of the presence of much earthy matter. Whenever the arsenic admits of solution in hydrochloric acid, however small the quantity present, it may be readily obtained as chloride. This distillation process has the advantage of not interfering with the search for mercury, lead, copper, and other poisonous metals which do not form volatile chlorides. Arsenic is thus separated from them, and these metals may be found in the residue contained in the flask or retort. Even antimony, which forms a volatile chloride, is not so readily distilled over as arsenic.

The distilled liquid may be at once submitted to a further analysis, any or all of the various tests being employed. A sufficient volume of the distillate (whose total volume must be measured) should be set aside for quantitative estimation (Marsh-Berzelius or Gutzeit method) if that is not done first.

Of course, if considerable amounts of arsenic are present, the estimation may be made by precipitating and weighing the sulphide.

The method of Fresenius and Babo is very useful for the destruction of organic matters and the obtaining of arsenic and other metals from organic mixtures, the solid organs, etc. The substance to be examined—if a solid, finely minced or ground—is placed in a porcelain dish and treated with a quantity of hydrochloric acid of the specific gravity 1·12, equal to, or rather exceeding, the weight of the dry substances present, and sufficient water to give the entire mass the consistence of a thin paste. The quantity of hydrochloric acid added should never exceed one-third of the entire liquid present, since if the acid is concentrated there is grave danger of losing part of the arsenic as chloride (which, it will be remembered, is volatile). Heat the dish on a water-bath, adding from time to time—say, every five minutes—two grains of chlorate of potassium for each fluid ounce of liquid in the dish, with stirring, until the contents of the dish are light yellow in colour, homogeneous, and fluid. A further addition of diluted hydrochloric acid may be requisite, when much chlorate is added, for the destruction of the organic matter. The operation is completed when the liquid after a fresh addition of either chlorate or acid does not deepen in colour when heated anew on the water-bath for a quarter of an hour. When this point is attained, add again a little chlorate and then cool the dish. When quite cold, filter, wash, and heat the filtrate on the water-bath, with renewal of the evaporated water, until all odour of chlorine, or nearly so, has disappeared. The precipitate on the filter paper will contain barium, silver and lead, if these were present. The chlorine may be eliminated rapidly by boiling the filtrate with potassium metabisulphite, after which excess of sulphur dioxide can be removed by bubbling a current of air through the hot liquid. The liquid thus obtained, measuring about thrice the bulk of the hydrochloric acid employed, is transferred to a flask and heated to from 150° to 160° F., and a slow stream of washed sulphuretted hydrogen gas is passed through it for twelve hours. The flask is then allowed to cool, the stream of gas being continued, and is then set aside (stoppered) overnight for precipitation to be completed. Any precipitate which forms is collected on a filter, and washed with water containing sulphuretted hydrogen till the washings are quite free from chlorides. The precipitate contains the arsenic and also any antimony, mercury, lead, tin, cadmium, bismuth, or copper which may be present, along with free sulphur. The filtrate may contain salts of aluminium, chromium, zinc, nickel, barium, etc. The precipitate is filtered off and heated on the water-bath with 10 per cent. hydrochloric acid and excess of bromine, heating until the whole of the bromine has disappeared. The liquid is then filtered, excess of a solution of stannous chloride is added and the liquid boiled. The Reinsch or Marsh test may now be applied to part of this liquid, and, possibly, arsenic may be estimated from the remainder by precipitation as sulphide.

The incineration method of Stryzowski depends on the formation of magnesium pyroarsenate, which is so slightly volatile that loss from that cause is negligible. The minced material (100 grams is usually a suitable amount) is mixed with one-third of its weight of a saturated solution of magnesium nitrate, and sufficient magnesium oxide to keep the reaction alkaline to litmus. [The magnesium compounds must, of course, be proved free from arsenic.] The mixture is carefully heated on a sand-bath or in an oven at about 250° C. until thoroughly dried—about three

hours. The grey, spongy mixture is then transferred from the porcelain basin in which it has been dried, to a silica basin, and is heated over a naked Bunsen flame or in a furnace at a dull red heat until incineration is complete—about half an hour, when a furnace is used. After cooling the white ash is redissolved in acid. When metals other than arsenic are sought, nitric acid is best, and the solution is evaporated to dryness, when the residue, redissolved in water, is available for testing. When it is a question of testing for arsenic, the ash is extracted with dilute sulphuric acid, and the filtered extract is used for the Marsh-Berzelius test, preferably after addition of a little stannous chloride.

Digestion with a mixture of sulphuric and nitric acids has been found by Dr. E. B. Hendry, working in the Editor's laboratory to be particularly convenient in dealing with small amounts of material. The minced tissue is heated with its own volume of concentrated sulphuric acid, and small amounts of pure fuming nitric acid are added at intervals of a few minutes until further addition of nitric acid and heating causes no darkening of the pale yellow liquid. The process is best started in a porcelain basin, but as soon as the mixture becomes fluid it may conveniently be transferred to a flask or boiling-tube. After destruction of the organic matter, excess nitric acid is boiled off after addition of a little water. The last traces are removed by boiling with a little sodium metabisulphite, which simultaneously reduces arsenic acid, and the solution is tested by the Marsh-Berzelius or Gutzeit methods.

The following references are useful :—

'Royal Commission on Arsenical Poisoning."
Heffter, "Ueber die Ablagerung des Arsens in den Haaren." *Vrtljschr. f. ger. Med.*, 1915, 49, 194–205.
Underhill, "The distribution of Arsenic in a Human Body." *J. Biol. Chem.*, 1914, 19, 513.

Cases. The following is quoted from *B.M.J.*, Epit., p. 21, for August 8th, 1903 :—

Methyl disodic arsenate was first introduced into therapeutics by A. Gautier in 1902, and is largely prescribed by French physicians under the name of *arrhénal*. It consists of white crystals having the formula $CH_3AsO_3Na_2H_2O_2$. It is freely soluble in water, sparingly soluble in alcohol, and insoluble in ether. Gautier recommends *arrhénal* in the place of the cacodylates, which are apt to disturb the gastric and intestinal canals. The usual dose for intestinal or subcutaneous administration varies between 0.025 gr. and 0.1 gr. per diem. A. Le Roy des Barres[1] places on record a case of acute poisoning by *arrhénal*. A patient, aged forty, was ordered a course of arsenic in the form of *arrhénal* in doses of 0.05 gr. per diem for a fortnight in each month, the cure lasting three months. At the same time Hunyadi Janos water was prescribed as an aperient. By a mistake on the part of the patient's attendant he was given the entire bottle of *arrhénal*, containing 1 gram. Five minutes later vomiting without pain commenced, followed in a few minutes by diarrhœa. Within twenty-four hours the patient vomited twelve times and passed twenty liquid stools. During the second day no vomiting occurred, but five or six diarrhœic stools were passed. No trouble was experienced after the fourth day, and the patient was observed for over a month to remain free from all secondary symptoms of poisoning. This case appears worthy of record for several reasons; first, cases of poisoning by this drug are extremely rare; secondly, they present marked differences from the ordinary cases of acute poisoning by other preparations of arsenic. In doses of 1 gram, vomiting (without hiccough) and diarrhœa only are present. There is an absence of burning acid sensation in the hypogastrium, of

[1] *Arch. Gén. de Méd.*, June 30th, 1903.

acute abdominal spasm, and of headache. The patient is free from loss of heat in the extremities, from muscular cramp, from collapse, and from cardiac syncope. Convalescence is uninterrupted, and the symptoms of reaction, such as fever, tympanites, jaundice, cutaneous eruptions, etc., which often herald a fatal termination in cases of acute arsenical poisoning, are completely absent. The author suggests that the benign course of the case recorded may be due to the exceedingly poisonous dose of the drug absorbed, producing vomiting and diarrhœa immediately; but he points out how different are the symptoms in non-fatal cases of acute poisoning by other arsenical preparations.

For fatal cases of arsenical poisoning by vaginal introduction, *vide B.M.J.* Epit., 1879, 1, No. 56; also 1899, 2, No. 9. Accidental case of arsenic in vinegar, *vide B.M.J.*, 1898, 1, p. 735; it was given to some forty or fifty soldiers, with acute symptoms. In the *Lancet*, 1, 1901, p. 1199, will be found an account of arsenical poisoning arising from stockings dyed with arsenical pigments, and a reference to cases occurring in 1897.

In May 1904, at Kendal, Elizabeth Nicholson and Thomas Metcalf were charged with poisoning James Gilpin by arsenical poisoning between March 17th and 20th, 1904. So far as motive and opportunity (given in gruel to an old bedridden man) and symptoms the case had no features of interest. The poison was openly purchased, and for the defence it was alleged that it had been taken by accident. At the final trial the prisoners were eventually acquitted. In Sir Thomas Stevenson's evidence there was one point of some importance, *viz.*, the fact that the arsenic was coloured with ultramarine. His evidence was as follows: that on April 7th he received three jars from the Chief Constable of Kendal. They were securely fastened and sealed with Dr. Hellon's seal. Two were marked "B" and "C," and the other one he marked "A." The box (produced) containing a portion of a rat-hole was also handed to him. Subsequently he received a packet of arsenic by registered post. He had carefully examined and analysed the articles. "A" jar contained the stomach of an adult person and weighed 8 oz. The mucous membrane showed signs of inflammation, and there were some small haemorrhages below the mucous membrane. There was no arsenic visible to the naked eye in the stomach. The "B" jar contained portions of the small bowel of an adult, which showed signs of acute inflammation. There were no particles of arsenic or pigment visible to the naked eye. The "C" jar contained three portions of bowel of an adult. The mucous membrane was inflamed. There was no arsenic or pigment visible to the naked eye in these contents. The appearances of the stomach and bowels were those of a person who had died from gastro-enteritis – inflammation of the stomach and intestines. They were in a good condition, considering the person had been dead a fortnight, which was suggestive of death from metallic irritant poisoning, not necessarily arsenic, which would act as a preservative. He determined the quantity of arsenic present at 0·689 of a grain. Dr. Hellon's quantities added to these made a total of 0·712 of a grain in the three jars. If the arsenic were taken through the mouth it must have been taken a considerable time before death—he should say many hours. It might have been given a couple of days before death. The effect of vomiting would be to remove arsenic from the stomach and the upper part of the small intestines. The packet produced contained over half an ounce of white arsenic, 83 per cent., coloured blue, with blue pigment of ultramarine. The presence of that particular pigment (the colouring) was very important. If it had been indigo or soot it would not have been affected so far as the colour went by the acid of the stomach. In the case of the arsenic coloured by ultramarine the acid of the stomach destroyed the colour. An ordinary fatal dose for a healthy adult would be 2 grains of white arsenic, or $2\frac{1}{2}$ grains of blue arsenic. He was of opinion that half that dose, or a little more, would kill an old and feeble man. The box containing the rat-hole contained rough plaster and partly burnt coal, weighing 9 oz. The whole of the arsenic in the contents of the box would not amount to $\frac{1}{10}$ part of a grain; he could find no trace of arsenic on the surface of the plaster. There was usually a trace of arsenic in plaster and coal, and what trace he did find was of a very insignificant quantity. On April 16th, by the licence of the Home Secretary, he attended Kentmere cemetery for the purpose of exhuming the body of James Gilpin. Most of the body was unusually well preserved considering that it had been buried for twenty four days. He examined all the chief organs, and did not find any appearance to account for the natural death of the deceased. The heart, lungs, and kidneys were quite competent to perform their functions. There

were only the usual appearances of degeneration met with in persons of middle and old age. He removed the following viscera for purpose of analysis: the whole liver (weight 37 oz.), kidneys (9½ oz.), spleen (3½ oz.), the heart and its appendages (19½ oz.), 4 oz. of fluid from the chest cavity, and muscle from the thigh (15½ oz.), also 42 grains of hair from the head. He reserved the heart, spleen and hair. In all the other organs he found absorbed arsenic to the extent of 0·195 of a grain. This was not a very large quantity, but quite sufficient to show that deceased had received a fatal dose. Taking the quantities given as the basis for the whole body, the quantity absorbed would be from 2 to 3 grains at the least, this being in addition to that in the stomach and intestines. From the quantities he had found, coupled with the symptoms during life and *post-mortem* after death, he had no doubt Gilpin died from acute poisoning by arsenic. From the evidence it would appear that it was an anonymous letter which led to police inquiries, the medical man who attended deceased having given a certificate of death without suspicion, the victim being described as an octogenarian.

Poisoning by Antimony

Source and Method of Occurrence. Antimony shares with arsenic the unenviable *rôle* of being the choice of the wilful poisoner, most of the *causes célebrès* of modern times having to do with one or the other, or both.[1]

Antimony in the form of tartar emetic is used medicinally in the shape of vinum antimoniale, strength two grains to the ounce (official dose, ten to thirty minims or two to four drachms as an emetic) and this is the form in which the metal is usually employed for criminal purposes. There are two chlorides of antimony, the trichloride and the pentachloride; the latter, butter of antimony, is also used as "bronzing liquid" for trade purposes, and occasionally cases of accidental poisoning occur from it. Taylor collected thirty-seven cases, of which fifteen were fatal.[2]

Tartar emetic [Potassium Antimonyl tartrate $2(KSbOC_4H_4O_6)H_2O$], in the state of powder, is white and crystalline. It has been occasionally sold by mistake for tartaric acid, with soda powders, and sometimes for cream of tartar. Its official dose is one-thirty-second to one-eighth grain as a diaphoretic, half to one grain as an emetic. It is commonly used for intravenous injection for bilharziasis in doses of one-half to two grains every second day for twelve doses.

Various organic antimony preparations have been put on the market in recent years for the treatment of tropical diseases. Amongst these are stibenyl, antimosan, Urea Stibemine, Bayer E.B. 212, stibosan, neostibosan, etc. Attention may be called to the use of antimony oxide in the manufacture of cheap enamel ware. The oxide may be dissolved by acid liquids placed in the vessels, and a number of cases of poisoning have been traced to this cause (p. 455).[3]

Toxicity and Fatal Dose. The smallest fatal dose of tartar emetic was in a child **three-quarters of a grain**, and in an adult **two grains**; but in these instances there were circumstances which favoured the fatal operation of the poison.[4] Although these very small doses with fatal results are on record, there are marked differences as regards severity of symptoms and fatality with much larger quantities; and in view of the fact that

[1] Binz, "Pharmacology," E. Tr., vol. 2, p. 318.
[2] Guy's Hosp. Rep., 1857.
[3] Ministry of Health, Memo. 171/Med., 1933.
[4] Guy's Hosp. Rep., 1857.

two grains are given intravenously as a routine dose for the treatment of bilharzia, these cases cannot be accepted as the normal. The differences probably depend in some degree on whether active vomiting and purging have been excited or not, for these symptoms have not been present in all cases. Doses of from twenty grains to one ounce have been taken without destroying life, although alarming symptoms of irritation have followed. In one case, a man, *æt.* 50, took forty grains of tartar emetic, and died in about four days. This was the only one out of five cases of poisoning by this substance quoted by Orfila which proved fatal.[1] Beck mentions a case in which fifteen grains of tartar emetic in solution killed a child in a few weeks ; vomiting and purging were among the symptoms, and these were followed by convulsions and death. Mr. Bravo also vomited freely, yet he died (see p. 447, *post*), which proves that a patient is not always saved by vomiting and purging ; but it must be remembered that in his case opium very probably shared in causing death. A dose of four grains, however, has been known to produce alarming symptoms.

A man, *æt.* 28, swallowed *two drachms* of tartar emetic by mistake for Epsom salts, and recovered from its effects.

A boy, *æt.* 12, swallowed by mistake for ginger-beer four or five drachms of a solution of butter of antimony. In half an hour he was seized with vomiting, which continued at intervals for two hours. Remedial means were adopted, and he recovered in about eight days. Another case of recovery from a dose of one ounce is reported.[2]

Duration. In acute cases death may occur within twenty-four hours ; but it is more common at a later period. An adult was killed in ten hours by a dose of one drachm in spite of early and violent vomiting.[3] In two cases *ten grains* killed each child in a few hours.

In 1881 a young man was killed in **six hours** by a dose of fifteen grains of tartar emetic. The characteristic eruption of tartar emetic was found on the mucous membrane of the stomach.[4]

A veterinary surgeon swallowed, by mistake for carbonate of sodium, about 200 grains of tartar emetic in powder. He noticed a peculiar taste. Vomiting came on in fifteen minutes, but only after tickling his throat. This continued violently. In two hours there was severe purging, with symptoms of collapse. The vomited matters were green, and the evacuations like boiled sago. There was no appearance of blood in either. In three hours severe cramps came on affecting all the muscles ; he was unable to move or speak. Brandy and other remedies were employed and in six hours, after a warm perspiration, he began to recover. There was suppression of urine ; only a small quantity was passed, and this was of a coffee colour. For two or three days he suffered from stiffness in the limbs and in the muscles of the abdomen. In one case, fifty-five grains caused the death of an adult in sixteen hours.

Two children, a boy aged five years and a girl aged three years, each swallowed a powder containing *ten grains* of tartar emetic. In twenty minutes after taking the powders, they were seized with violent vomiting and purging, and great prostration of strength, followed by convulsions and tetanic spasms : there was also great thirst. The boy died in eight hours, and the girl in twelve or thirteen hours, after swallowing the dose.

[1] Orfila, vol. 1, p. 480.
[2] *Lancet*, February 26th, 1848, p. 250.
[3] *Med. Gaz.*, vol. 45, p. 801.
[4] Friedreich's *Blätter f. Gerichtl. Med.*, 1882, p. 8.

A girl, *æt.* 16, swallowed a dose of tartar emetic amounting to from forty to sixty grains. There was severe vomiting in a quarter of an hour, and this was soon followed by purging; these symptoms continued for about three hours. She also complained of pain and a burning sensation along the gullet. The vomited matters were of a dark colour. On the following morning she had recovered from the severity of the symptoms; but in the afternoon there was a relapse. She continually threw her head back and screamed; the skin was warm and moist; the pupils were dilated, and the knees drawn up. She died in about thirty-six hours after taking the poison, and during the six or eight hours previous to her death she was delirious.

In another case an army surgeon swallowed, for the purpose of suicide, from two to three fluid ounces of chloride of antimony. About an hour afterwards there was entire prostration of strength, with coldness of skin, and incessant attempts to vomit. The most excruciating griping pains were felt in the abdomen; and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was now a strong disposition to sleep, so that he appeared as if suffering from the effects of a narcotic poison. In this state he continued until he died, ten hours and a half after he had swallowed the poison.

Symptoms. When *tartar emetic* is taken in a poisonous dose, a strong metallic taste is perceived in the mouth during the act of swallowing. In from a few minutes to an hour there is great heat and constriction of the throat, with difficulty of swallowing, violent burning pain in the region of the stomach, followed by incessant vomiting and, later, profuse purging, faintness, and extreme depression. The symptoms are indeed those of acute gastro-intestinal inflammation. The pulse is small, rapid, and sometimes imperceptible; the skin cold, and covered with a clammy perspiration; and the respiration is painful. Should the case prove fatal death may be preceded by giddiness, insensibility, great prostration of strength, and sometimes violent spasms of the muscles of the extremities, which may assume either a clonic or a tetanic character. Such are the symptoms in an acute case of poisoning by this substance. Cases of poisoning by tartar emetic at first greatly resemble those of poisoning by arsenic; but in fatal cases the remissions often seen in arsenical poisoning are absent, and recovery is common even when there is great collapse. Occasionally a pustular eruption like that produced by the external application of tartar emetic appears on the skin.

After intravenous injection the common symptoms observed are coughing, nausea, vomiting and temporary giddiness. A metallic taste is often noticed. Collapse occasionally occurs and rarely sudden death. Persistent vomiting, diarrhœa, cramps and diminution of urine are symptomatic in some patients.[1]

Proctor met with the cases of four children to whom, in 1860, by mistake, a mixture of sulphur and tartar emetic had been given. An ounce of sublimed sulphur and one drachm of tartar emetic had been divided among the four. The symptoms presented the same characters in each: early vomiting, which became violent and incessant, pain in the bowels, purging, great thirst, cold clammy perspiration, feeble pulse, cramps of the limbs and twitchings of the muscles, with great depression. There was no sense of heat or constriction in the throat, and no difficulty of swallowing. Under treatment they all recovered.

A man, *æt.* 28, swallowed two drachms of tartar emetic, and in an hour after the poison had been taken he was found in the following state:—His pulse was imperceptible, the tongue was dry and red, the

[1] Khalil, *Archiv. für Schiff und Tropen-Hygiene*, 1926, Band 30, S. 451-467.

countenance cold, livid, bathed with clammy perspiration, and indicative of great suffering; violent pain was felt in the stomach and over the whole of the abdomen, with constant spasmodic contraction of the muscles, particularly of the abdomen and arms. The fingers were firmly contracted, and the muscles quite rigid. He vomited once, about half an hour after he had swallowed the poison; and after this he had constant involuntary watery purging. An emetic of mustard and salt was given to him, and this produced violent vomiting of bilious matter. Green tea, brandy, and decoction of oak-bark were freely given. The cramps, vomitings and watery purging continued for six hours. The symptoms then became mitigated, and he gradually recovered, suffering chiefly from profuse night sweats.[1] This case is remarkable for the unusual character of the symptoms, as, in the absence of active vomiting, it was necessary to give an emetic, and also for the recovery of the patient after so large a dose of the poison.

On April 18th, 1876, Mr. Bravo, a barrister, *æt.* 30, was poisoned by tartar emetic. After dining with his wife and whilst alone in a room, at 6.30 p.m., he was suddenly seized with violent sickness and vomiting. When seen at 10.30 p.m. by Moore, he was lying back in a chair totally unconscious; the breathing was noisy, and the heart's action was barely perceptible. He did not seem to suffer pain, and his appearance was not unlike that of a person under the effects of a narcotic. He had previously complained of pain in the stomach, and an emetic of mustard and water had been given. The pupils were widely dilated; and he was unable to swallow when seen shortly afterwards by Harrison. At 1.45 a.m. he first vomited blood. At 3.30 a.m. on the 19th, soon after he was seen by George Johnson and Royes Bell, a little consciousness returned; and he then said, to account for his state, that he had rubbed his gums with laudanum, and that he might have taken some of this liquid. Just before becoming unconscious—*viz.*, at 2.45 a.m.—he vomited blood, and passed bloody stools. Throughout the 19th, after the return of consciousness, he suffered excruciating pain, and was violently purged and vomited frequently. On the 20th the patient was seen by Gull, at 6.30 p.m. who found him pulseless and dying. He was told that he was dying of poison, and was asked how he came by it. He replied, "I took it myself." "What did you take?" "Laudanum." When told that he had taken more than laudanum, he made no further statement bearing upon the matter, except to say a second time: "I took it myself." Pain, collapse, vomiting, purging, and occasional convulsions, especially of the upper limbs, continued till near the close, when the vomiting and purging ceased. He died on the 21st of April, fifty-five hours and a half after the commencement of symptoms.[2]

The *chloride* gives symptoms practically identical, but with more local pain and signs of irritation.

In 1868, a man swallowed three or four ounces of bronzing liquid, which proved to be a solution of chloride of antimony. He vomited violently, but continued his work for an hour; the vomited matters were of a yellow green colour. There was pain in the stomach, but no purging. He was not seen by a medical man. He had passed a sleepless

[1] *Lancet*, May 22nd, 1847, p. 535.
[2] *The Times*, 1876, July 12th, etc.

night, and complained much of oppression in the region of the heart. He died in about eighteen hours.

Four grains gave rise to violent pain in the abdomen, vomiting, and purging. The patient then fell into strong convulsions, which lasted half an hour. He became speechless, no pulse could be perceived, the skin was cold, and it was supposed that he was dead. Stimulating frictions and poultices were employed, and he slowly recovered in about fourteen days.

Borgzinner (quoted by Leschke, "Clinical Toxicology," 1933, p. 76) reports the following fatal case of poisoning by *antimosan*.

A woman, aged 42, was treated with antimosan for multiple sclerosis in 1926. Seventeen injections (2·35 grams in all) were given, with a good measure of success, over a period of two months. In 1927 the injections were renewed, but after the third (at an interval of five days) she collapsed in the consulting room, with pains in the sacral and stomach regions. A few hours later she vomited and coughed up blood, afterwards becoming deeply somnolent, with pale, cyanosed lips. The reflexes could not be elicited, there were extensive purpural hæmorrhages on the trunk and extremities, as well as in the conjunctivæ and in the mucous membrane of the mouth. The pulse was weak and hurried, bronchitis developed, the liver was swollen, the urine contained a good deal of albumen with leucocytes, and there was a leucocytosis of 12,800. *Post-mortem :* purpura, marked fatty degeneration of the liver, softening of the spleen, pneumonia, flabbiness and enlargement of the heart.

Chronic Poisoning. Criminal poisoners rarely resort to a single large dose of antimony for their purpose ; they more commonly give small doses at intervals.

The principal symptoms are those of gastro-intestinal catarrh, *viz.*, nausea and vomiting of mucous and bilious liquids ; great depression ; watery purging, often followed by constipation ; small and frequent pulse ; loss of voice and muscular strength ; coldness of the skin, with clammy perspiration ; and death from exhaustion. Several cases have occurred in this country which show that tartar emetic has been thus criminally employed. In addition to the cases of Ann Palmer and J. P. Cook, there are those of *R.* v. *M'Mullin*[1] ; *R.* v. *Freeman*[2] : and the cases of the James family at Liverpool.[3] The prisoner Winslow was indicted for the murder of his mistress Ann James. It was clearly proved that antimony had been administered to the deceased, not only from the symptoms, but by the detection of the poison in the urine during life. The deceased was at the time labouring under malignant disease affecting the cæcum and stomach, but it was alleged that her death had been accelerated by antimony. The prisoner was acquitted, owing to the difficulty of proving the act of administration. The poison had been given at intervals in small doses, and as deceased survived about a fortnight after the last dose, it was found only in traces in the various organs. The death of this woman led to the exhumation of the bodies of three of her relatives who had lived in the same house with her and the prisoner, and had died suddenly some months previously in suspicious circumstances. The viscera of these bodies were examined, and in each case antimony

[1] Liverpool Sum. Ass., 1856.
[2] Drogheda Spring Ass., 1857.
[3] *R.* v. *Winslow*, Liverpool Aut. Ass., 1860.

was found in small quantity, but still extensively diffused through the organs. So far as the history of their cases could be obtained, they were victims of chronic poisoning by antimony. This cause of death was not suspected at the time.[1]

Administered in small doses, antimony may cause death by its depressing influence on the heart. Aged persons, or those who are debilitated by disease, may die in these circumstances from a medicinal dose or doses which would produce no injury to strong and healthy adults. The effects, however, should be clearly traced to the action of the poison, and not be owing to exhaustion as a result of disease. In 1853, a case was referred to Taylor in which it was supposed that two doses of antimonial wine, equal to about *three grains* of tartar emetic, had caused the death of a man who was in a diseased condition by its remote effects upon the heart. No trace of poison was found in the stomach or tissues; there were no symptoms to indicate poisoning, so that death could not be reasonably attributed to the medicine. The man died in about twenty hours, probably from exhaustion of the vital powers as a result of disease, and not from the action of this substance. This shows that medical men should be alive to the possibility of crime in such circumstances as ill health.[2]

Antimony, formerly much employed in febrile conditions, is now seldom used except for intravenous injection in anthelmintic work, and its presence in a body on analysis is very suspicious.

Treatment. If vomiting has not occurred, it should be induced by means of an emetic of mustard and water, or by tickling the fauces with a feather, after which the stomach tube may be employed to wash out the stomach. In cases of poisoning by "butter or chloride of antimony" the stomach tube should not be used. After the stomach has been emptied a drachm of tannic acid, dissolved or suspended in warm water, may be administered, with the object of forming an insoluble tannate of antimony. Opium should be given to relieve the pain, and ice may be administered to allay the vomiting and gastric irritation. Demulcent drinks, such as linseed tea, almond mixture and milk, may be taken, and warmth and stimulants should be employed, if necessary, to overcome the depressing action of the poison.

Post-mortem Appearances. The preparations of antimony are irritants, and the chloride is a corrosive. Hence possibly corrosion, and certainly irritation, of the stomach, are to be looked for; but it is exceedingly unlikely that the eye will reveal in any special way that antimony was the cause of the irritation. In the case of the boy and girl above, the bodies were examined between four and five days after death. In that of the boy there was effusion of serum in the right pleura; the lower lobe of the right lung posteriorly was redder than natural, and the peritoneum was injected from recent inflammation. The mucous membrane of the duodenum was inflamed, and covered with a whitish yellow viscid secretion; this was observed throughout the intestines, although the colour was of a deeper yellow in the large intestines: there was no ulceration. The peritoneal coat of the stomach was inflamed.

[1] See Guy's Hosp. Rep., 1857 and 1860; also *R.* v. *Hardman*, Lancaster Sum. Ass., 1857, pp. 369–481.

[2] Vide also Caillol de Poncy et Livon sur l'empois, chronique par l'antimoine, *Compt. Rend. Acad. d. Sc.*, 1882, 95, 695.

The mucous membrane of this organ was also much inflamed, especially about the larger curvature and at the cardiac orifice; but there was no ulceration. The contents (about two ounces and a half of a dark bloody fluid having a slightly acid reaction) were adherent to it; and in one case there was a patch of lymph. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur, and appeared sodden; the throat was not inflamed; the windpipe and gullet had a natural appearance. On opening the head the dura mater was found congested; the longitudinal sinus contained a coagulum. The vessels of the surface of the brain were much injected with dark blood, the whole surface having a deep purple colour. Every portion of the brain, when cut, presented many bloody points. The cerebellum and medulla oblongata were also congested: there was no effusion in the ventricles or at the base of the brain. In the body of the girl the morbid appearances were similar; there were in addition patches on the arms, legs, and neck resembling the eruption of scarlatina. The arachnoid membrane was more opaque than usual; and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration.[1]

At the autopsy made on Mr. Bravo the day after death the following appearances were observed by Payne. There was no sign of inflammation, congestion, nor ulceration. The stomach contained about eight ounces of thick, gruel-like matter, of a yellowish colour, containing small solid lumps, and had the odour of alcoholic fermentation. The gullet was natural, and contained some of the same matter as the stomach. The first part of the bowels was so soft that it was torn in tying it, but subsequent careful examination showed no perforation nor ulceration. The surface was pale and yellowish, like that of the stomach. The whole of the small intestines was like this, except the lower part, where there were some red spots. This part of the bowels contained yellow pasty matter, without any admixture of blood. Subsequent examination showed, in the cæcum, several small ulcers from which the bleeding had evidently taken place, but there was no perforation. The remainder of the large intestine was very deeply blood-stained, but without ulceration. The contents were soft dark-red material, composed of fæcal matter mixed with blood. The liver and spleen were natural, as were also the pancreas, kidneys, and other abdominal organs. The skull and the membranes of the brain were found quite natural, containing only the usual amount of blood. The brain-substance was also natural, and contained no excessive amount either of blood or of watery fluid. The mouth and lips were natural, except that the papillæ at the back of the tongue were somewhat more prominent than is usual. There was no other appearance of disease in the body, except what had been noted.

In the case of the girl *æt.* 16 (above), an inspection was made thirty-six hours after death. The throat appeared swollen: the lungs were slightly congested; the heart was healthy, and contained about six drachms of fluid blood. The stomach contained sixteen ounces of a thick bloody fluid; at the greater extremity the coats were softened, and blood was effused under the mucous coat in several places. The small intestines

[1] *Lancet*, April 25th, 1846, p. 460.

contained a similar fluid, with much mucus ; but there was no appearance of inflammation. Only slight traces of the poison were found in the contents of the stomach by the usual tests, the greater part having probably been lost by vomiting and purging.[1] In animals poisoned by this substance, Pavy, and the author found general inflammation of the lower half of the intestines.

In a case where antimony chloride had been used, the interior of the alimentary canal, from the mouth downwards to the jejunum, presented a black appearance, as if the parts had been charred. In general, there was no mucous membrane remaining, either on the stomach or elsewhere ; only a flocculent substance, which could be easily scraped off with the back of a scalpel, leaving the sub-mucous tissue and the peritoneal coat. All these parts were so soft that they were easily torn by the fingers. On further inspection, the mucous membrane of the stomach was found much corroded. Near the intestinal end there were numerous putty-like masses. In parts it was a vivid red colour, and in other parts blackened. There was no perforation. The duodenum presented similar appearances. There was no mark of corrosion on the lips, nor in the lower part of the gullet. The upper part of this tube, the fauces and mouth, could not be examined. Antimony was found in the putty-like masses of membrane as well as in the contents of the stomach and the liquid swallowed.

Analysis—General Remarks. The general remarks on p. 426 with regard to arsenic apply equally well to antimony. It must be remembered that the discovery of antimony in the contents of a stomach is by no means a proof of its having been taken or administered as a poison, since tartar emetic is sometimes prescribed as a medicine, although seldom taken as such by persons of their own accord. We could only suspect that it existed as a poison, or had caused death, when the quantity present was large and there were corresponding appearances of irritation in the stomach and bowels. The presence of any quantity, if not lawfully administered as a medicine, is always a suspicious fact and demands explanation. In two cases of criminal administration in small doses, the quantity found in each body did not exceed three grains. The mere discovery of it in a medicinal mixture cannot of itself be evidence of an intent to poison.

The detection of antimony in the tissues does not necessarily indicate that it has been criminally administered nor that it has caused death ; but its presence should be reasonably accounted for, as antimony may have been unlawfully administered. At the same time it must be remembered that antimony is not a natural constituent of the body, nor are minute traces of it so commonly found in food materials as may be, the case with arsenic or copper. In several cases of suspected death from poison, deposits on copper, evidently of an antimonial nature, have been obtained from the liver or tissues. On inquiry it has been found that antimonial medicines had been taken shortly before death. Conversely, when no antimony is found, or the metal is present in the tissues in minute quantity, it is still consistent with medical experience and observation that the person may have died from antimony. The case of Mrs. Peters, of Yeovil (July 1860), furnishes a remarkable illustration of this fact.

[1] *Lancet*, 1854, 1, p. 68.

This lady had symptoms during her illness which were referred by her medical attendants to the effects of small doses of antimony. Antimony was found in the urine by them as well as by Herapath ; but after death (*i.e.*, in about nine days) no antimony was found in the tissues nor any part of the body. Upon this fact, and the evidence of co-existing disease, it was alleged that she had died from disease and not from poison. The jury returned a verdict to the effect that her death had been accelerated by irritant poison.[1]

The case is important in this respect : it shows that antimony may be found in an evacuation, and that death may be accelerated by the metal ; but although the person may die within nine days, none may be detected in the body.

From the first vomit of Mr. Bravo, Redwood obtained antimony equivalent to ten grains of tartar emetic. Antimony was also detected in the liver, and in fluid taken from the large intestines of the deceased, but not in the contents of the stomach. Traces of the metal were also discovered in the urine passed during life. Redwood was of opinion that at least twenty grains of tartar emetic had been taken by the deceased. A verdict of wilful murder by some unknown person was returned, no tartar emetic being traced into the hands of the deceased nor of any other person about him. It was also known that Mr. Bravo was well read in medical jurisprudence, and was acquainted with the poisonous nature of tartar emetic. Some months after the return of the above verdict, evidence came into the possession of Sir Thomas Stevenson, who watched the medical aspect of the case at the inquest on behalf of one of the persons living in the same house as the deceased, showing that Mr. Bravo had tartar emetic in his possession within six or seven weeks of his decease. He had purchased a large number of quack powders, extensively advertised as a cure for dipsomania, and received instructions that these, if administered too freely to his wife, would cause vomiting. It is possible that these powders—each of them consisting of half a grain of tartar emetic with a pink vegetable pigment—may have been taken in fatal amount by the deceased, who was unaware of their poisonous nature, for the purpose of causing ejection of the laudanum which he had admittedly taken. It is probable that the laudanum would delay the ejection of the tartar emetic, and thus increase the liability to a fatal result.

Chemical Analysis. As in the case of arsenic, the Reinsch test may be used as a preliminary means of detecting small amounts of antimony in organic fluids, tissues, etc., but for confirmation of the result of this test by quantitative analysis, or for the detection of minute amounts too small to give definite results by the Reinsch test, it is necessary to destroy the organic matter by one or other of the methods described in the section on arsenic.

Hydrogen sulphide bubbled through a solution of an antimony salt, or passed over a solid, produces the characteristic orange-red antimony sulphide. This precipitate is insoluble in cold dilute hydrochloric acid and in ammonium hydroxide. It is, however, dissolved in boiling concentrated hydrochloric acid, in sodium and potassium hydroxide solutions, and in a solution of ammonium sulphide. Its colour distinguishes it from other metallic sulphides, but can only be relied upon in the absence of organic matter.

[1] *Med. Times and Gaz.*, 1860, 2, pp. 190, 271 and 317.

Water, in excess, converts antimony chloride to the insoluble oxychloride. The sulphide precipitated from the test solution by hydrogen sulphide is filtered off and dissolved in hot hydrochloric acid. Careful addition of sodium hydroxide to this solution causes precipitation of antimony oxide after the excess of acid has been neutralised. [This precipitate is easily missed, as it dissolves in excess of the alkali.] The precipitate is then redissolved, without filtration, in the minimum amount of dilute hydrochloric acid, and a drop or two of the resultant solution is poured into a beaker of water. A white precipitate indicates the presence of antimony or bismuth. The two are readily distinguished by the fact that antimony oxychloride is soluble in tartaric acid, whereas bismuth oxychloride is not.

Zinc. The suspected liquid, strongly acidified with hydrochloric acid, is placed in a platinum dish and a zinc rod is immersed in it, touching the dish. Antimony, *but not arsenic*, is deposited on the platinum as a brown or black stain. The identity of the stain may be established by dissolving it in nitric acid, and adding hydrogen sulphide solution, when orange antimony sulphide is precipitated. The antimony deposit may also be obtained by immersing in the acidified liquid (contained in a glass or porcelain vessel) a piece of platinum-foil wrapped round with zinc-foil. The liquid should not be so strongly acid as to attack the zinc rapidly. The test may be applied in the presence of organic matter. One ten-thousandth of a grain gives a distinct stain.

Tin. Metallic antimony is deposited from acid solution on tin-foil immersed in the solution. Arsenic is not deposited.

Reinsch's Test. The technique of this test has already been described (p. 430). In the case of antimony the deposit on the copper is reddish or violet at first or if the amount of antimony is small. Larger amounts of antimony form a grey stain with a metallic lustre, or a black amorphous layer. For identification, the antimony may be removed by solution in dilute potassium permanganate, or the dried copper strip may be heated in a glass tube open at both ends, when antimony trioxide is formed and sublimes on the cool part of the tube. The sublimate—usually amorphous—may be dissolved in hydrochloric acid for testing further, or hydrogen sulphide may be passed over it, when it is converted to orange antimony sulphide.

Marsh-Berzelius Test. The deposit on the heated tube in this test (see p. 432 for technique) may be identified as antimony by the following tests :—

(1) The deposit is grey or black (not brown) and is often found on both sides of the heated part of the tube.

(2) Heat the deposit cautiously after blowing air through the tube to remove hydrogen. A white sublimate in the cool part of the tube is usually amorphous in the case of antimony, whereas that from arsenic consists of octahedral or tetrahedral crystals.

(3) If a stream of hydrogen sulphide is passed over the metallic deposit or the sublimate of oxide, the non-volatile orange sulphide is formed from antimony, the volatile canary-yellow sulphide from arsenic.

(4) The metallic deposit of antimony, unlike that of arsenic, does not readily dissolve in a solution of calcium hypochlorite (bleaching powder).

Gutzeit Test (see p. 435).

Quantitative Estimation. For small amounts, the Marsh-Berzelius method may be used, a series of standard deposits being prepared as in the case of arsenic. The Gutzeit method may also be employed, again with a freshly prepared series of standard stains.

Larger amounts may be estimated as sulphide as follows:—The solution obtained after destruction of organic matter is acidified with hydrochloric acid (if not already acid) and saturated with hydrogen sulphide. The flask is stoppered and allowed to stand for two hours, and the precipitate is then filtered off and washed. [Precipitate A.]

(*a*) If lead, copper, mercury, or bismuth[1] may be present, the precipitate A is extracted two or three times with yellow ammonium sulphide solution. The extracts are acidified with hydrochloric acid and re-saturated with hydrogen sulphide. The precipitate is filtered off and washed. [Precipitate B.]

(*b*) If arsenic[2] may be present, precipitate B (or A if lead, etc., are known to be absent) is boiled with concentrated hydrochloric acid until the smell of H_2S is no longer perceptible. The mixture is then diluted with water and filtered. The filtrate contains antimony, while arsenic sulphide remains insoluble. Antimony sulphide is then precipitated by saturating the solution with hydrogen sulphide. [Precipitate C.]

The precipitate [A if antimony alone is present, B if arsenic is absent but lead, copper, mercury, or bismuth may be present; C if arsenic may be present as well] is transferred to a weighed porcelain or silica boat which is introduced into a horizontal combustion tube through which a gentle stream of dry carbon dioxide is passed. The boat is then heated to expel water and sulphur. It is allowed to cool, the current of gas being continued, and is then weighed again. The heating is repeated until the weight is constant. The *black* antimony sulphide which remains in the boat contains 71·77 per cent of antimony.

Cases. (1) An extraordinary trial for murder by alleged poisoning with this substance took place at Annapolis, U.S., in 1871. Mrs. Wharton was charged with poisoning her friend General Ketchum. The General died after a short illness, but the symptoms, taken as a whole, bore no resemblance to those observed in poisoning with antimony, although poisoning was suspected during life. The appearances in the body proved nothing for or against antimonial poisoning and some physicians of experience deposed that the symptoms and appearances were consistent with disease affecting the membranes of the brain and spinal marrow. On examining the chemical evidence, it appears that sulphide of ammonium alone was employed for the detection of antimony, and a red-brown sulphide resembling that of antimony was obtained; but the quantity obtained as sulphide was only four-tenths of a grain, estimated as equivalent to *eight-tenths of a grain* of tartar emetic. Thus the chemical analysis brought out only a fraction of a grain, not amounting to one-twentieth part of the quantity said to be present; and no separation of antimony in the metallic state was made to corroborate the inference drawn from the coloured precipitate produced by sulphuretted hydrogen. No chemical results were produced in court, although the amount found would have allowed the production of metallic antimony in a few minutes by copper, tin, zinc, and

[1] Jones (*Lancet*, January 23rd, 1926) has shown that so-called pure tartar emetic may contain traces of lead and arsenic.

[2] *Lancet*, January 23rd, 1926.

platinum, or by Marsh's process. The evidence that antimony was really there was not satisfactory, and that twenty grains were present in the stomach was wholly unproved. The chemical evidence does not therefore conflict with the pathological evidence, for it failed to show with clearness and distinctness the presence and proportion of the poison said to have been found. The accused was acquitted.[1]

(2) In *R.* v. *Matchett*, Armagh Assizes, March 1904, it was stated that the tartar emetic was bought for dosing cattle. Happily for the victim, the poison was put into the tea in such an excessive quantity that when drunk it had the effect of causing him to become violently ill and to vomit. The man managed with assistance to get away to the house of his brother, and was there put to bed. His condition became critical, and he was attended by a doctor for some time. He recovered, and gave evidence that the tea was bitter to taste, and when his sister-in-law's attention was withdrawn he threw the remains of it away. He was subsequently taken violently ill, accompanied by vomiting.[2]

(3) Two women, mother and daughter, were tried[3] for the murder of the illegitimate infant of the daughter by means of terchloride of antimony. They were acquitted because there was not sufficient evidence of administration. It was at first supposed that the child had died from poisoning by a solution of perchloride of iron, which was detected in the stomach by a medical man. Sir Thomas Stevenson found antimony in the viscera, and also in the matters vomited and passed from the bowels. The perchloride of iron was merely an impurity always present in commercial liquid butter of antimony.

(4) In the summer of 1928, a firm in Newcastle-on-Tyne provided its employees with cooling drinks made from "lemonade crystals" which were contained in enamelled buckets and allowed to stand overnight. By midday 70 employees had helped themselves to the lemonade; nearly all were sick, and 56 had to be taken to hospital. The symptoms consisted of a burning sensation in the stomach, colicky pains, nausea, vomiting, and collapse. Intestinal symptoms were absent. Recovery was rapid; only two patients were detained overnight in the hospital; and no sequelæ were recorded. A tumblerful of the lemonade contained antimony equivalent to 1½ grains of tartar emetic, and the enamel of the buckets contained about 3 per cent. of antimony oxide.

Similar outbreaks occurred at Folkestone in 1929, and in London in 1932, and in both cases the poisoning was caused by lemonade made from fresh fruit and contained in enamelled vessels.[4]

Poisoning by Bismuth Salts

Source and Method of Occurrence. The carbonate and the subnitrate of bismuth are extensively used in medicine, as are also the oxide and the salicylate.

It has been found that bismuth salts are opaque to the X-rays, and consequently emulsions of bismuth salts are given in large doses in order to examine patients on a screen for certain affections of gullet, stomach, and intestine, and also to determine certain points in the process of digestion.

In oral administration so little of the metal is absorbed that toxic effects are rarely seen. Many of the cases reported in which toxic symptoms have arisen after large doses of bismuth subnitrate are not cases of bismuth poisoning, but of poisoning by nitrites.

In these cases the nitrate has been reduced to nitrite by the action of putrefactive bacteria, and the absorption of the nitrite has produced cyanosis, methæmoglobinæmia, dyspnœa and death by arrest of respiration. A case is recorded[5] in which nitric oxide hæmoglobin was found in the blood.

[1] *Amer. Jour. Med. Sc.*, April 1872, p. 329.
[2] *Vide* also *R.* v. *Klosowsky*, p. 342, *ante*.
[3] *R.* v. *Wallis and Wallis*, Worcester Sum. Ass., 1883.
[4] Ministry of Health, Memo. 171/Med., 1933.
[5] *Presse Méd.*, February 25th, 1920.

W. H. Resnik[1] reports a case of bismuth poisoning in a woman suffering from diabetes mellitus. Before admission to hospital she had been given in a fortnight about five to seven ounces of bismuth subnitrate by the mouth. The symptoms comprised a bluish-black discoloration of the gums, which were swollen and inflamed; a similar discoloration of the tongue, most noticeable at the apex of the papillæ and arranged in vertical striations along the lateral margins; a patchy, diffuse discoloration of the buccal mucosa; swelling and tenderness of the parotid glands; a moderate anæmia, the erythrocytes being 3,100,000 per cubic millimetre, the hæmoglobin 40 per cent., and the leucocytes 9,000 per cubic millimetre; and basophilic stippling of the red cells. The clinical picture, in fact, closely resembled that of lead poisoning. Bismuth was detected in the urine. Recovery followed the withdrawal of the salt.

The use of bismuth injections for the treatment of syphilis has increased considerably in the past few years with a corresponding increase in our knowledge of its toxic effects.

Bismuth preparations are from ten to twenty times more toxic when injected intravenously than when injected intramuscularly.

The absorption of bismuth has been studied especially by Christiansen and others[2] by a radio chemical method. They consider that its absorption is slow and irregular, and that there is a risk of cumulative effects. They say that it is eliminated chiefly by the kidneys and to a less extent by the bowel, contrary to the usual belief.

The excretion by the urine commences within eighteen to twenty-four hours of the first injection, and has been found as long as three months after the last injection.

The common toxic symptoms observed are stomatitis, which may be preceded by a slaty blue line along the gums, nephritis and enteritis. Toxic effects on the central nervous system have also been described. Hudelo and Rabat[3] discuss the toxicology at some length. They describe gastro-intestinal symptoms, such as diarrhœa and vomiting, jaundice, pruritus, and various skin eruptions which may occasionally develop into severe exfoliative dermatitis.

O. Fischer[4] describes a case of severe bismuth poisoning after a course of intravenous injections of bismuth-diasporal, a preparation which has been extensively advertised as having a low degree of toxicity. A man, aged twenty-six, was treated by this drug. Four injections of 50 mgrm. each were given in the course of ten days. In the following week, two injections, each of 100 mgrm., were given, all without toxic symptoms, but after the sixth injection the patient had pain in the lower teeth and a "bismuth line" around the lower incisors. The seventh injection was postponed for five days; but immediately after this the patient complained of pain at the site of the injection and the whole arm became stiff, apparently owing to phlebitis. The patient had severe attacks of abdominal pain and diarrhœa, followed by blood-stained, mucous stools. He had also severe hæmaturia, and other signs of acute nephritis, such as albuminuria and epithelial casts. The abdomen was very rigid, especially in the left flank. Proctoscopic examination was painful and revealed an

[1] *Bull. Johns Hopkins Hosp.*, May, 1926, p. 232.
[2] *Comptes Rend.*, April 7th, 1924.
[3] *Presse Méd.*, April 9th, 1924. Reviewed in the *Medical Annual*, 1925, p. 441.
[4] *Dermatol. Woch.*, February 20th, 1926, p. 268.

extensive ulceration of the mucosa. The enteritis and nephritis improved, and four weeks later the patient was discharged.[1]

We quote the following from the *Lancet*, 1909, vol. 1, p. 257 :—

Bismuth Subnitrate Poisoning. Until recently bismuth has been regarded as non-toxic, and the few recorded cases of poisoning after its use have been ascribed to the presence of arsenic or other impurity. But it has now been shown that pure bismuth has toxic properties, and its recent administration for purposes of radiography has brought this fact more into evidence. In the *New York Medical Journal* of January 2nd Dr. Emil G. Beck has published an exhaustive paper on the subject. As long ago as 1793 cases of poisoning by bismuth subnitrate were recorded, but were thought to be due to impurities. The first authentic report of bismuth poisoning was published by Theodor Kocher in 1882. He observed that insoluble preparations of bismuth when applied to large wound surfaces may be absorbed to such an extent as to produce characteristic symptoms—acute stomatitis and black discoloration, usually beginning on the gums at the border of the teeth and spreading over the whole mouth, which are followed by intestinal catarrh, pain, and diarrhœa, and in severe cases by desquamative nephritis.[2] Similar cases were reported by Professor Petersen.[3] Then literature was silent on the subject until 1901, when Professor Muhlig reported two cases of poisoning after the dressing of burns with bismuth subnitrate. Later two cases of poisoning were reported in America after administration of 60 and 120 grams respectively of the subnitrate in one dose. The first fatal case was reported by Bennecke and Hoffmann.[4] An emaciated child, aged three weeks, suffered from enteritis. A mixture of three grams of bismuth subnitrate in 100 cubic centimetres of buttermilk was given in order to diagnose pyloric stenosis by radiography. Twelve hours later cyanosis developed and was followed by collapse and death. After death methæmoglobin and small quantities of bismuth were found in the liver and blood. In the same clinic another fatal case with similar symptoms occurred in a child. Professor Hefter suggested that the symptoms might be due to nitrite poisoning. The blood and pericardial fluid were tested for nitrates, which were detected in both. Then Böhme proved by experiments that the fæces of children in contact with bismuth subnitrate liberate nitrites. He injected this mixture into a ligated loop of a rabbit's intestine and found nitrates and nitrites in the urine, but not in the blood. Routenberg has reported a case in which methæmoglobinæmia and the other symptoms of nitrite poisoning followed the rectal injection of 50 grams of bismuth subnitrate in 400 cubic centimetres of oil of sesame.[5] These and other cases show that toxic effects from bismuth subnitrate may be due to absorption not of bismuth, but of nitrites. The liberation of nitrites appears to be due to the action of bacteria in the alimentary canal, especially in the sigmoid flexure and rectum. During the past two years Dr. Beck has treated sinuses extensively by injection of a paste containing 33 per cent. of bismuth subnitrate incorporated in petrolatum.[6] If the bismuth was not discharged soon after injection he proved by a series of radiograms that it underwent absorption. In no case did he observe symptoms of nitrite poisoning, but in many cases he noticed slight lividity of the skin, of the mucous membranes, and a bluish border on the gums. In one case he observed ulceration of the mouth, which has been described by Kocher as characteristic of bismuth poisoning. In another case he injected 720 grams of the paste into a pleural cavity from which an empyema had been evacuated. The paste was retained for six weeks, and then desquamative nephritis and a blue border around the teeth developed. The bismuth paste was withdrawn by injecting olive oil and applying a specially devised suction pump, and all the symptoms disappeared. Recently Dr. H. Eggenbergel has reported a fatal case of bismuth intoxication following injection of the sinus of a psoas abscess in a child, aged seven years,[7] from the clinic of Professor Wilms, of Basle. Thirty grams of the paste were injected and retained for six weeks. Stomatitis resembling that due to mercury developed, the pulse rose to 130, and

[1] *B.M.J.*, April 3rd, 1926.
[2] Volkmann's *Klinische Vorträge*, p. 224.
[3] *Deutsche medizinische Wochenschrift*, June 20th, 1883.
[4] *Münchener medizinische Wochenschrift* 1906, No. 19.
[5] *Berliner klinische Wochenschrift*, 1906, No. 43, p. 1397
[6] *Lancet*, May 9th, 1908, p. 1359.
[7] *Centralblatt für Chirurgie*, October 31st, 1908.

cerebral symptoms like those of uræmia followed. The abscess cavity was evacuated, but death occurred a few days later. It thus appears that two forms of poisoning may be produced by bismuth subnitrate—an acute form due to nitrite poisoning and a more chronic form due to bismuth poisoning.

Treatment. The only treatment is that applicable to poisons in general, evacuation of the stomach and bowel, attention to the mouth and kidney and general symptomatic treatment. Sodium thiosulphate has been recommended; see "Arsenic Poisoning."

Post-mortem Appearances. Skin lesions and ulceration of the buccal mucous membrane may be observed.

There may be signs of gastro-enteritis and occasionally ulceration, especially in the region of the cæcum and colon. The cæcum may appear quite black, and this colour may extend through the whole thickness of the wall of the gut. The kidneys show signs of acute nephritis.

Analysis. The subnitrate, carbonate and oxide of bismuth are white chalk-like powders, insoluble in water, but soluble in dilute nitric or hydrochloric acid. The nitrate and chloride are crystalline, but form amorphous powders (oxy-salts) when added to water. They dissolve in dilute nitric or hydrochloric acid.

Solutions of bismuth salts form a dark-brown or black precipitate on addition of hydrogen sulphide or ammonium sulphide (the solid substances are also blackened by these reagents); they give a white precipitate with sodium hydroxide, insoluble in excess of the reagent; they give (most easily when the minimum of free acid is present) a white precipitate when added to a large volume of water; and they are not precipitated by dilute sulphuric acid. Minute traces of bismuth in dilute acid solution (1 mgrm. bismuth per 100 cc. of solution) give an intense yellow colour on addition of a 10 per cent. solution of thiourea; the test is said not to be interfered with by the other metals of group II (lead, mercury, cadmium, arsenic, antimony, tin, etc.).[1]

Arsenic may be detected in bismuth salts by dissolving in slightly diluted hydrochloric acid and introducing the solution into Marsh's apparatus. The arsenical flame is apparent on combustion, and the usual deposits may be obtained on glass and porcelain. The products of combustion may be collected and tested by the processes described for arsenic. This impurity in the subnitrate may modify a conclusion respecting the presence of traces of arsenic in a body when bismuth has been administered medicinally.[2]

Bismuth in Urine. As the estimation of small quantities of bismuth in the urine may be of considerable importance, the following method devised by Hill[3] is given:—

To Destroy the Organic Matter. As the amount of bismuth is usually unknown, and as it is desirable to carry out the final colorimetric matching in a solution containing about 0·05 mgrm. in 50 c.c., three separate quantities of 5, 20, 50 c.c. each of the urine should be measured into separate silica beakers of 100 c.c. capacity and evaporated on an asbestos board over a Bunsen flame to about 5 c.c.; the beakers are removed and 5 c.c. of specific gravity 1·4 nitric acid added, the basins replaced on the asbestos and evaporated to dryness and heated at the lowest possible

[1] *Annali Chim. Appl.*, 1929, 19, 392.
[2] *Brit. and For. Med. Chir. Rev.*, October 1858.
[3] *Lancet*, December 19th, 1925.

temperature, so that the organic matter is oxidised but not charred; it is important to stop the ignition when all the nitric acid is driven off and then to remove, cool, and moisten with more nitric acid; again, the evaporation and heating are repeated with previously mentioned precautions and if necessary the procedure repeated with further small quantities of nitric acid until a white residue remains. After cooling, two drops of nitric acid and 25 c.c. of distilled water are added and the solution boiled for a few minutes and transferred to a Nessler cylinder; if the lowest temperature for the destruction of the organic matter has been maintained, it will be unnecessary to filter the solution, which should be perfectly clear and colourless. After rinsing out the beaker with further quantities of boiling water, the volume is made up to 50 c.c. and cooled.

To Determine the Bismuth. To the solution in the Nessler cylinder add 0·3 gram of urea, dissolve, and mix thoroughly, then add 0·5 gram phenazone, agitate to dissolve, and while still shaking add potassium iodide in very small crystals (average weight 0·05 gram) one at a time until a red colour appears; it will be readily seen when the maximum colour develops, then add one crystal in excess. The colour is quite easily imitated with a standard solution of bismuth made by dissolving 0·62 gram of bismuth carbonate B.P. in 10 c.c. nitric acid (specific gravity 1·4), adding water to 1,000 c.c., and further diluting 10 c.c. of this to 1 litre. The best results in the control experiment are obtained by taking 10 c.c. of this solution (equivalent to 0·05 mgrm. of bismuth, Bi) and 1 drop HNO_3 (specific gravity 1·4), adjusting to 50 c.c. in the Nessler cylinder and adding the reagents as the method specifies, and the comparison is made with the solution giving the most suitable depth of colour or in a colorimeter.

Test results to show the accuracy of the method were made by adding known amounts of bismuth to varying quantities from different samples of urine and treating as described. In every experiment the colour obtained was strictly comparable with the standard containing the same amount of bismuth. Four different samples of urine into which no bismuth was introduced failed to produce any colour when similarly treated.

Cases. *Bismuth Subnitrate—Pearl White—Magistery of Bismuth.* This substance, in a dose of 120 *grains*, caused the death of an adult in nine days. There was a strong metallic taste in the mouth, burning pain in the throat, with vomiting and purging, coldness of the surface, and spasms of the arms and legs. On inspection, the throat, windpipe and gullet were found inflamed; and there was inflammatory redness in the stomach and throughout the intestinal canal.[1] In a case mentioned by Traill, a man took by mistake *six drachms* of the subnitrate in divided doses in three days. He suffered from vomiting and pain in the abdomen and throat.[2] These cases are sufficient to prove that a substance which is but slightly soluble in water may exert a powerfully poisonous action. The oxide and subnitrate of bismuth, owing to imperfect washing, are sometimes contaminated with arsenic in the form of arsenic acid; and as the symptoms produced by large doses have closely resembled those caused by arsenic, the symptoms may have been due to this impurity.

REFERENCES

Mayer and Baehr, "Bismuth Poisoning," *Surg. Gynæcol. and Obstet.*, Chicago, 1912, 34, 868.

Higgins (*Jour. Amer. Med. Assoc.*, 1916, 66, 648) reviews the literature on bismuth poisoning.

[1] "Sobernheim," p. 335.

[2] "Outlines," p. 115.

Poisoning by Iron Salts

Source and Method of Occurrence. Iron salts are amongst the most valuable drugs in the pharmacopœia, and there are very many pharmacopœial preparations of them. The sulphate and the perchloride are the two which form the basis of most of these preparations, and it is from these two salts that cases of poisoning may arise.

Ferrous Sulphate, or Sulphate of Iron—Copperas, or Green Vitriol. This compound has been several times administered with malicious intention. One death from it took place in 1837–8, and another in 1869 was the subject of a criminal trial in France. A man was convicted of having killed his wife and his son by administering to them ferrous sulphate in coffee.[1]

Green vitriol or copperas is sometimes given as an abortifacient, and in this connection the tincture of the perchloride is advertised largely under the name of steel drops.

A suspicious case is reported in which a woman far advanced in pregnancy, but enjoying good health, was at midnight suddenly seized with vomiting and purging, and died within about fourteen hours. The body, which had been buried, was disinterred, and iron found in large quantities in the viscera (*vide* also "Abortion").

Copperas is said to have proved fatal to sheep. It has been mixed with the pulp of beetroot for cattle food.[2]

Ferric Chloride—Solution of Perchloride of Iron. The solution of perchloride of iron of the British Pharmacopœia contains about 15 per cent. of $FeCl_3$, and the official dose is five to fifteen minims.

Perchloride of iron has been used as an injection in uterine diseases; but it is a most powerful local irritant, and in one instance caused death by inducing peritonitis. The mucous membrane of the uterus was stained a deep black colour, and iron was readily detected in its substance.[3] The solution of ferric chloride in water is usually definitely acid since the salt is partly hydrolysed with liberation of free hydrochloride acid, and this may account, in part at least, for its irritating effect.

A case was reported to the Westminster Medical Society, in 1842, in which a girl, *æt.* 15, five months advanced in pregnancy, swallowed *an ounce* of the tincture of perchloride of iron in four doses in one day, for the purpose of inducing abortion. Great irritation of the whole urinary system followed, but she recovered.

At the Lincoln Lent Ass., 1863,[4] a druggist was convicted of having supplied this noxious liquid to a woman with the intent to procure her miscarriage. He directed her to take a teaspoonful three times a day, and at the same time prescribed for her eight pills a day, each containing half a grain of powdered cantharides. Although the woman had taken only two doses of the tincture of perchloride of iron, she suffered from severe pain over the whole of the abdomen, with violent pain in the region of the stomach and bladder; there was constant vomiting of a greenish-coloured matter, and great pain in passing her urine. The quantity of urine secreted was small, and contained much blood. These symptoms were in great part attributable to the cantharides.

It is commonly asserted that pregnant women are affected injuriously by comparatively small doses, but there seems to be no evidence that

[1] Bouchardot, "Ann. de Thérap.," 1872, p. 146.
[2] *Med. Times and Gaz.*, 1863, 1, p. 511.
[3] *Amer. Jour. Med. Sci.*, April 1870, p. 506.
[4] *R.* v. *Rumble.*

they are so affected by any dose between the pharmacopœial limits, which were largely exceeded in the above case.

Toxicity and Fatal Dose. The toxicity of iron salts is undoubtedly very slight, and it is difficult to find a fatal case. A girl swallowed an ounce of the sulphate and recovered.

Christison relates an instance in which a man by mistake swallowed *an ounce and a half* of the tincture of the perchloride: the symptoms were somewhat like those produced by hydrochloric acid. He at first rallied, but died in about five weeks. The stomach was found inflamed and thickened towards the intestinal end. In another case, a healthy married woman swallowed, by mistake for an aperient draught, *one ounce and a half* of the tincture of perchloride of iron. She immediately ejected a portion, and violent retching came on, which continued for some time. There was great swelling of the glottis, with cough and difficulty of swallowing. These symptoms were followed by heat and dryness of the throat, with a pricking sensation along the course of the gullet and stomach; and in the afternoon a quantity of dark liquid blood was vomited. The motions were black, owing, doubtless, to the combination of sulphur with the metal. Within about a month the patient was perfectly restored to health.[1] Another case of recovery from a large dose has been reported. The patient, *æt.* 72, swallowed by mistake *three ounces* of the tincture in a concentrated state. The tongue soon became swollen; a ropy mucus flowed from the mouth and nose; there was croupy respiration, and a sense of impending suffocation. The pulse was feeble, the skin cold and clammy, and the face swollen and livid. A castor-oil mixture brought away inky evacuations, and the patient rapidly recovered.[2] The largeness of the dose has commonly led to early vomiting, and the rejection of the greater part of the acid liquid. Besides, it varies much in strength, and unless this is defined in any given case it is difficult to draw an inference of the actual quantity of perchloride of iron taken.

Symptoms. Pain in the stomach, vomiting and purging are likely to be found in an acute case.

In 1879, a teaspoonful of the stronger pharmacopœial solution of perchloride of iron was given, undiluted, to a girl, *æt.* 18, by mistake for a linctus. The bowels were freely opened, and she vomited several times within ten minutes of taking the liquid. The stomach was then washed out. Shortly after she again passed a stool, and continued to vomit freely. The nurse and patient both asserted that vomiting and purging supervened within three minutes of the administration of the poison. Great feebleness and depression came on. In two days she began to recover; but pain in the gullet continued for several days (unreported case of Sir Thomas Stevenson).

A case of recovery from an ounce of this tincture is reported.[3] A woman, *æt.* 30, swallowed this quantity. She suffered from vomiting and purging, the motions being black. Emetics were given, and she recovered within five days.[4]

[1] *Prov. Jour.*, April 17th and 21st, 1847, p. 180.
[2] *Dub. Med. Press*, February 21st, 1849.
[3] *Pharm. Jour.*, April, 1869, p. 605.
[4] *Lancet*, 1869, 1, p. 9.

It must be specifically noted that all the symptoms referable to poisoning by iron are concerned with the immediate irritating effects upon the alimentary canal. Iron being a natural and very essential constituent of the body, there is no suggestion of any symptoms due to the absorption of iron from the stomach or elsewhere ; it thus contrasts very strongly with lead, arsenic, barium, etc., which are not constituents of the body.

Treatment. But little is required beyond the general measures on pp. 250 *et seq.* Bicarbonate of soda might be useful to form a less soluble iron salt. The case can them be treated as simple gastritis.

Post-mortem Appearances. If any, will be those of gastro-enteric irritation.

Analysis. The hydrochloric acid of the perchloride may be detected by nitrate of silver and nitric acid, while the iron is immediately indicated by a precipitate of Prussian blue on adding a solution of *ferrocyanide of potassium*. If it be a ferrous salt that is present, the precipitate, on adding potassium ferrocyanide, is at first nearly white, but changes to a light blue ; potassium ferrocyanide, however, gives an immediate deep blue precipitate. As iron is a natural constituent of the body, it is obviously useless to discover mere traces of the metal in the viscera, and generally, it is necessary to show quantitatively that abnormal amounts are present.

Cases. At the Nottingham Autumn Ass., 1859, a woman named Riley was prosecuted for administering copperas to two children. She put the substance into gruel. It gave to the gruel a greenish colour and a peculiar taste which led to the discovery. It caused sickness, but no other serious symptoms. As there was no evidence of an intent to murder, and it was then not unlawful to administer poison with any other intent, the accused was acquitted.

Poisoning by Osmium

Osmium tetroxide, commonly known as osmic acid, is a highly poisonous substance, and accidents have occasionally resulted from its use in scientific investigations. It is a transparent, glistening, crystalline body, melting like wax in the hand, beginning to sublime at a moderate heat, and boiling at 212° F. It does not redden litmus, and has a caustic, burning taste. Its odour is most penetrating, and somewhat like that of chlorine or of iodine. The vapour, even in minute quantities, causes serious inflammation of the lungs when inhaled. It also attacks the eyes, so that the experiment of volatilising osmium in the blowpipe is an operation attended with great danger. Deville, who performed the operation twice, was rendered almost blind for twenty-four hours by having become exposed accidentally to the vapour of the tetroxide. This substance produces violent pain and inflammation of the conjunctivæ ; and vision may be permanently injured by the subsequent production of a film of metallic osmium. The tetroxide also acts violently on the skin, causing painful eruptions.

Poisoning by Chromium

Source and Method of Occurrence. Chromium is largely used for "plating," and the chromates and bichromates of various metals are manufactured in very large quantities for industrial use ; but fatal cases of poisoning by it are not common.

Solutions used in certain electric batteries, consisting of a mixture of the bichromate and sulphuric acid, are practically solutions of chromic and sulphuric acids. In 1892 a man committed suicide by swallowing a battery fluid of this nature.

The workers in chrome factories have been found to suffer to such an extent in health that this trade has been scheduled under the Workmen's Compensation Acts, *vide* Vol. I.

Toxicity and Fatal Dose. Two drachms of potassium bichromate have proved fatal in four hours; and Leschke[1] records the suicide of a woman who died on the tenth day after swallowing 15 grams of potassium bichromate.

Chromate of Lead (chrome yellow) is a powerful irritant poison: a dose of a few grains of this pigment has proved fatal.

Chromic acid is a powerful corrosive poison destroying all organic textures.

Symptoms. Bichromate usually acts as a powerful irritant. In the first two hours a woman suffered from violent vomiting and purging, the vomited matters being of a yellow colour. When admitted she was in a dying state, pulseless, unconscious, and breathing slowly with great effort. The skin was cold, the lower lip swollen and purple, and the tongue swollen. Occasionally these irritant symptoms are not present, and the drug acts by its effects on the nervous system after absorption (*vide* cases below). In cases of acute poisoning, vomiting and diarrhœa are early symptoms. Later there are signs of severe kidney damage—oliguria with albuminuria and hæmaturia, or even anuria. The victims complain of headache, stiffness of the neck, cramps, etc., and the pupils are dilated.

This salt, in the state of fine powder or in a saturated solution, has also a local irritant action on the skin and on parts from which the skin has been removed.[2] It produces what are called "chromic sores," affecting the hands and exposed parts of the face. According to recent observations, workers in chrome factories—of which there are very few in the world—suffer from a peculiar irritation and ulceration (chrome holes) of the septum of the nose, apparently due to the action of bichromate of potassium, or more probably chromic acid set free, which leads to a perforation of the nasal septum. Ulcers of the septum were found in 107 and perforation in 87 out of 237 workmen. Mitchell[3] published the results of the examination of 846 workers in chrome extending over three years; 20 per cent. of these men showed chrome sores. There are no specific appearances about these ulcers, but they must be distinguished from those due to syphilis, lupus and tubercle.

Treatment. Must be on general principles (*vide* pp. 250 *et seq.*): there is no specific antidote. Preventive measures are adopted in workshops.

Post-mortem Appearances. In the case mentioned above the chief appearances were: a dark and liquid state of the blood: the mucous membrane of the stomach was in great part destroyed, and was dark

[1] Leschke, "Clinical Toxicology," 1934, p. 85.
[2] "Ann. d'Hyg.," 1864, I, 83.
[3] *Jour. State Med.*, 1916, 24, 18.

brown approaching to purple: the duodenum at its upper part was a florid red colour, and at its lower part much corrugated, as was the upper half of the jejunum.

In addition, a yellow, red or greenish discoloration of the skin has been seen associated with parenchymatous degeneration of the kidney, liver, and cardiac muscle.

Analysis. Bichromate of potassium may be recognised by its orange-red colour, as well as by the colour which it gives to water when dissolved. Its solution gives a deep red precipitate with nitrate of silver, a pale yellow with nitrate of barium, and a bright yellow with salts of lead.

If a dilute solution of potassium bichromate be acidified with sulphuric acid, and sulphurous acid then added, a green colour is produced, due to the formation of chromium sulphate. A similar effect is produced by substituting alcohol for sulphurous acid, and subsequently warming the mixture.

The Perchromic Acid Test. The suspected liquid is acidified with sulphuric acid and treated with hydrogen peroxide. A blue colour due to the formation of perchromic acid is produced, and on shaking with ether the blue colour is transferred to the ether layer.

The Diphenyl-carbazide Test. A drop of the solution suspected of containing a chromate is acidified with dilute sulphuric acid and treated with a drop of a 0·2 per cent. solution of the reagent in a mixture of nine parts of alcohol and one part of glacial acetic acid. Chromate gives a violet colour.

Chromium salts in solution give the colour after being oxidised to chromates. This can be accomplished by bromine water, the excess bromine then being removed by making the mixture adhesive and adding a little phenol.

Cases. A man, *æt.* 64, was found dead in his bed twelve hours after he had gone to rest; he had been heard to snore loudly during the night, but this had occasioned no alarm to his relatives. When discovered he was lying on his left side, his lower limbs being a little drawn up to his body: his countenance was pale, placid and composed: the eyes and mouth closed: the pupils dilated: no discharge from any of the outlets of the body: no marks of vomiting or purging, nor any stain upon his hands or person, nor upon the bed-linen or furniture. The surface was moderately warm. Some dyestuff, in the form of a black powder, was found in his pocket. On inspection the brain and its membranes were healthy and natural: there was neither congestion nor effusion in any part. The thoracic viscera were healthy, as well as those of the abdomen, with the exception of the liver, which contained several hydatids. A pint of a turbid, inky-looking fluid was found in the stomach. The mucous membrane was red and vascular, particularly at the union of the greater end with the gullet: this was ascribed to the known intemperate habits of the deceased. In the absence of any obvious cause for death poison was suspected; and on analysing the contents of the stomach, they were found to contain bichromate of potassium. The dye-powder taken from the man's pocket consisted of this salt mixed with cream of tartar and sand. It is remarkable that in this case there was neither vomiting nor purging. The salt does not appear to have operated so much by its irritant properties, as by its effects on the nervous system. [It seems doubtful whether death was actually due to the chrome salt. Alcohol may have had a share in it.] This, however, is by no means an unusual occurrence, even with irritants far more powerful than bichromate of potassium.[1]

[1] *Med. Gaz.*, vol. 33, p. 734.

A boy recovered from the effects of a dose of bichromate of potassium, but only after a lapse of four months. The first symptoms were pain, vomiting, dilated and fixed pupils, cramps in the legs, and insensibility. His recovery was due to early and active treatment.[1] In another case, owing to timely treatment, a man, *æt.* 37, recovered from a large dose of the salt. It seems that with suicidal intent the man swallowed about two ounces of the bichromate in solution, mixed with pearlash. In about two hours he was seen by Andrews, and he was then apparently in a dying state. He was suffering chiefly from severe cramps, the pupils were dilated, the pulse was scarcely perceptible, and there was vomiting and purging of greenish-coloured evacuations. The stomach-pump was used, and olive oil and diluents were given. In about nine hours the urgent symptoms abated, and the man complained only of great pain in the shoulders and legs. There was no gastric irritation nor tenderness of the abdomen. He was discharged cured at the end of a week.

A fatal case occurred at Liverpool in which a woman, *æt.* 21, obtained and swallowed as a cure for neuralgia a quarter of an ounce of bichromate of potassium. She took it on the advice of an ignorant person who had written the name of the drug on paper as "Bichromide," and the chemist dispensed bichromate instead of bromide, which was the original intention of the adviser. The husband mixed the quantity advised by his brother-in-law, and gave it to his wife, without reading the label. He first tasted it and found it very bitter, and the deceased said, "Never mind if it will cure me," and drank it eagerly. She at once complained of sickness and burning, and commenced to vomit. The doctor was called in, and on his advice the woman was removed by her husband in a cab to the Stanley Hospital, where she expired at six o'clock the following evening. Verdict at inquest: misadventure.

In the *Pharm. Jour.* December 19th, 1896, p. 542, is an account of an inquest held on a woman, *æt.* 29, who took "a spoonful" out of "two-pennyworth" of the salt. A few minutes later she complained of violent pains in the stomach, and commenced vomiting. Her husband prepared an emetic of mustard and water, but she refused to take it. The salt was taken about 10 p.m., and the woman died about 4 p.m. next day. Dr. Howie found her suffering from great collapse twelve hours after the dose was taken. She was livid, and her pulse was rapidly failing. At the inquest Dr. Howie said that she had died from "asphyxia and paralysis of the brain centres." Verdict, misadventure.

A case of suicidal poisoning by chromic acid is reported in the *Pharm. Jour.* March 17th, 1894, p. 791. The acid was kept to supply a battery.

In the *Trans. of the Med.-Leg. Soc.* for 1909–10 Willcox reports an interesting case of poisoning by bichromate of potassium. The symptoms were not observed. There were no gross naked eye changes in the viscera except slight brown discoloration of the wall of the stomach, which Willcox admitted might have been taken for *post-mortem* changes. On analysis, chromium was found in all organs.

ADDITIONAL REFERENCES

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Poisoning by Nickel

Source and Method of Occurrence. Nickel is used industrially for hardening steel and for nickel plating. Nickel carbonyl $Ni(CO)_4$ discovered by Mond, Langer and Quincke, prepared by passing a current of CO over finely divided metallic nickel, is a clear pale straw-coloured liquid, volatilising at room temperatures. By its soot-like smell it can be recognised in dilutions of 1 : 200,000 of air; the Bunsen flame becomes luminous when nickel carbonyl is present in the air to the extent of 1 : 400,000. When the Mond process of preparing pure nickel was first introduced in 1902 twenty-five men were poisoned by this substance, of whom three died. Workmen in nickel works commonly fall victims to

[1] Guy's Hosp. Rep., 1850, p. 216.

"nickel eczema," which is marked by intense itching œdematous exanthema and miliary vesicles.

Poisoning by nickel carbonyl has been scheduled as a disease of occupation under the Workmen's Compensation Act.

Symptoms. In two of the above fatal cases these were as follows :

Both men were ill for several days before their death, and their symptoms were similar to those exhibited by other men who had been attacked in the previous months but who had recovered. The most prominent features in all the cases were headache, giddiness, high fever, and rapid breathing. *Post-mortem* examination in two fatal cases revealed a fatty and in one instance a dilated heart.

Post-mortem Appearances. To the naked eye there is apparently nothing actually characteristic, but the pathology of the condition, according to Armit, who has investigated the subject experimentally, is as follows. Grossly the condition is one of pure poisoning by nickel. In detail this occurs by the following means and routes :—

In the lungs nickel carbonyl is dissociated and a compound, probably the hydrated subcarbonate, is deposited on the respiratory surface, from which it is dissolved by the tissue fluids and is then taken up by the blood. Some of the nickel finds its way directly through the lymphatic channels into the bronchial glands. In the dissolved condition the nickel enters into complex combination with some constituent of the body. The metal is carried by the blood to the tissues, but a selective absorption is exercised by the brain and adrenals. In some cases the lungs exert a specific selection, but the nickel only stays for a short time in these organs. The specific pathological changes produced are primarily a degeneration of the endothelial cells of the capillary vessels. It is possible that some further primary action is exercised on the ganglion cells in the brain and on the parenchyma cells of the adrenals. Hæmorrhages follow as the result of the fatty degeneration of the vessel walls, and secondary changes result from the effects of the hæmorrhages. Nickel is excreted by the kidneys and intestines. Both cobalt and iron carbonyls act similarly, the latter, however, being less toxic than the other two.

The presence of carbon monoxide in the blood has also been described.

Tests for Nickel. (1) An alcoholic solution of dimethyl-glyoxime added to a dilute solution of a nickel salt made faintly alkaline with ammonia gives a red crystalline precipitate.

(2) A drop of the solution to be tested is placed on a filter paper and exposed to ammonia vapour. A single drop of a 1 per cent. alcoholic solution of dithio-oxamide (rubeanic acid) is then added. In presence of nickel a blue colour is produced.

Poisoning by Cobalt

Cobalt is used in the manufacture of certain steels, and its salts are widely used in the preparation of blue pigments.

Among workmen in cobalt mines bronchial catarrh is common, and the "Schneeberg lung cancer" is a well characterised condition traceable to the cobalt ores of the Schneeberg and Joachimsthal mines. Since, however, these ores contain arsenic and radio-active metals, it is possible that their peculiarly poisonous effects must be ascribed chiefly to these impurities and especially to their radio-activity.

Cobalt poisoning has, however, been described as the result of inhaling ore dust which was entirely free from arsenic. The victim recovered, but suffered from hæmaturia for at least three months after the appearance of the acute symptoms—violent abdominal pain, and vomiting.

Test: To 2 c.c. of the solution to be tested add 1 gram of sodium acetate and 2 c.c. of a 0·5 per cent. aqueous solution of nitroso-R salt ($C_{10}H_4.OH.NO(SO_3Na)_2$): boil: add 1 c.c. of concentrated nitric acid, and again boil for one minute. Cobalt gives a permanent red colour. Iron also gives a red colour which, however, is destroyed by the boiling with nitric acid.

Poisoning by Uranium

The compounds of this metal are now largely used in the arts, and cases of poisoning by them have occurred. Indeed, it has been proposed to place them in the list of poisons officially current in Russia. Uranium salts cause severe inflammation of the gastro-intestinal mucous membrane and of the kidneys, and are distinguished from other metallic poisons by acting directly on the walls of the blood-vessels, and also by rendering the blood reducible with difficulty. In this respect they resemble prussic acid.

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Poisoning by Radium and other Radio-active Metals

Radium and its analogues are exceedingly poisonous—hence the care and circumspection with which their therapeutic use must be undertaken. It has been calculated that as little as 0·01 mgrm. of radium, distributed over the whole body, would be sufficient to cause death.

Industrially, radio-active salts are used in the manufacture of luminous paints. While most patent remedies, cosmetics, etc., advertised as containing radium are probably harmless, a number undoubtedly do contain traces of radio-active salts, and are potential poisons. Attention has already been called (p. 466) to the possibility of radium poisoning among workmen in mines dealing with ores which are primarily sought for the sake of other metals; it is obvious that the possibility must exist also among those engaged in the mining and extraction of radium itself.

Gettler and Morris (*J. Amer. Med. Assoc.*, 1933, p. 400) record a death from the ingestion of radio-active matter.

The patient, a man 52 years of age, was admitted to hospital suffering from emaciation, anæmia, and necrosis of the jaw. During the preceding five years he had drunk 1,400 bottles of a tonic water "Radiothor." Each bottle contained 60 c.c. of the water, and about 2 micrograms of radio-active substance. During the patient's stay in hospital of a few months, his expired air was found to be radio-active. Four weeks before death, part of the necrotic jaw was removed by operation. The emaciation and anæmia increased continually, and at death the patient weighed only 90 lbs. (weight at time of admission, 110 lbs.). The cause of death was necrosis of the jaw, abscess of the brain, secondary anæmia, and terminal broncho pneumonia. The *post-mortem* findings were: necrosis of the

jaw-bones, swollen kidney cortex, cerebral abscess of the right temporo-sphenoidal lobe, moderate coronary sclerosis, moderate nodular sclerosis of the aorta, and marked hyperplastic bone marrow (regenerative type). The heart, liver, lungs, spleen, kidneys, and portions of the femur, vertebræ, ribs, jaw-bone, and teeth, were examined for radio-activity. The weighed samples were ashed, and the ashes tested by electroscopic and photographic methods. It was calculated that the entire body contained 73·66 micrograms of radium, of which the skeleton contained 73·27, and the soft tissue only 0·39 micrograms.

The most important series of cases of industrial radium poisoning is that which occurred among girls in a New Jersey factory who were employed in painting the dials of clocks and watches with luminous paint. This paint, a preparation of zinc sulphide, contained traces of radium or mesothorium.[1] Among 800 girls employed between 1917 and 1924, five deaths have been proved to be caused by radium poisoning, and the same cause is strongly suspected in ten further deaths. It seems likely that other deaths may also have been caused by radium, and that other workers may have suffered permanent or temporary harm. The signs of radium poisoning appeared in girls who had worked steadily in the factory for a year or more. The symptoms consisted of general weakness, more or less severe progressive anæmia, necrosis of the jaw, and various crippling bone lesions. In some cases death was the result of a secondary infection leading to general sepsis. During life, the expired air of certain of these cases was found to be radio-active (by the electroscope) and *post-mortem*, both electroscopic and photographic methods demonstrated the presence of radio-active matter in the phagocytic cells of the reticulo-endothelial system and, especially, in the bones. In these cases the poison had been ingested owing to the girls' habit of "pointing" their paint-brushes in their mouths, and consequently, little or no radioactive matter was found in the lungs. In the case of a physicist, however, who worked continuously with luminous paint, and inhaled the emanation and radio-active dust (besides being exposed to penetrative emanation), the lungs were found, *post-mortem*, to contain 1 microgram of radio-active matter, while the skeleton contained 14 micrograms.

Similar cases have been reported from a Connecticut factory in which luminous paint was used.[2]

The course of the poisoning may be illustrated by the case of Dr. von Sochocky, a former technical director of the New Jersey company, who died in 1928 of an aplastic or regenerative anæmia.[3] From 1913 to 1920 he had extracted about 30 grams of radium from the ore, and had been exposed to intense penetrative radiation from radio-active material, and to the inhalation of radio-active dust. Since 1921 he had occasionally been exposed to external penetrative radiation from radium used for therapeutic purposes. He had an extensive radium dermatitis of the fingers of both hands, considerable jaw necrosis with buccal lesions, and was very anæmic at times. The anæmia and buccal sepsis were combated by hygienic measures, and visits to high altitudes. In June 1925 his expired air was found to be strongly radio-active—more so than that of any of the employees. His health was fair, and he was able to work until

[1] *J. Amer. Med. Assoc.*, 1925, 85, 1769; 1929, 92, 466.
[2] Flinn, "Laryngoscope," 1927, 37, 341; *Boston Med. & Surg. J.*, 1928, 197, 1309.
[3] Maitland, *J. Amer. Med. Assoc.*, 1929, 92, 466.

August 1928, when he became extremely weak, pale, and dyspnœic. Blood examination showed: hæmoglobin 28 per cent.; erythrocytes, 1,720,000; leucocytes, 4,000 with 48 per cent. polymorphonuclears; blood platelets, scant; practically no reticulocytes; red cells regular and showing no polychromatophilia. For the remaining three and a half months of life the clinical picture was typical of that of the terminal stage of aplastic anæmia. He was kept alive by thirteen blood transfusions at intervals of three to eight days, and in amounts of 450–900 c.c. In spite of the low platelet count there was no bleeding (apart from a fine purpuric rash over the extremities) until the end. Terminally, the leucocytes varied from 1,800 to 2,500 with very few polymorphs. On one occasion the icteric index was 6·8, and the indirect van den Bergh test was slightly positive. Just before death he developed a septic broncho-pneumonia, retinal hæmorrhages, hæmoptysis and hæmaturia. Treatment, including the extended use of raw and cooked liver and of liver extracts, was without effect.

Poisoning by Manganese

Source and Method of Occurrence. Manganese is normally present in traces in healthy living tissues. Reiman and Minot[1] found from 0·005 to 0·020 mgrm. of manganese per 100 grams of normal human blood.

Chronic manganese poisoning is of some industrial importance, and is caused by inhalation of the dust of manganese dioxide (pyrolusite) or other manganese compounds (*e.g.*, silicates) by those who handle, mill, or smelt the ore. Manganese compounds (chiefly the dioxide) are used in the manufacture of ferromanganese, manganese steel, dry batteries, chlorine, etc., and in the glass and pigment industries.

Symptoms. Nervous and psychical symptoms predominate. The earliest signs are muscular weakness, tingling of the extremities, and unsteadiness of both hands and feet. Walking becomes more and more difficult, the gait resembling that in paralysis agitans. There may be propulsion and retropulsion, tremor in writing, slurred speech, and mask-like face. The tendon reflexes are active. Mental changes may occur moderately early[2]—impulsive laughter and weeping, diminution in memory and concentrating power. The sexual power is decreased.

Pathology. Leschke[3] states that the liver is damaged, as shown by lævulose and galactose tolerance tests, and that, *post-mortem*, atrophy of ganglion cells is found, especially in the globus pallidus, putamen and nucleus caudatus, along with degeneration of Forel's bundle. Casamajor[4] also found, *post-mortem*, nephritis, cirrhosis of the liver, and degeneration in the fibres of the pons.

Treatment. The prognosis in chronic manganese poisoning is bad, so far as complete recovery is concerned. Some improvement usually follows removal of the victim from exposure to manganese-containing dust. The use of liver therapy has been suggested.

Analysis. Organic matter is removed by incineration, and the ash is dissolved in dilute hydrochloric or sulphuric acid.

[1] *J. Biol. Chem.*, 1920, 42, 329.
[2] von Jaksch., *J. Amer. Med. Assoc.*, 1913, 61, 1042.
[3] "Clinical Toxicology," 1934, p. 80.
[4] *J. Amer. Med. Assoc.*, 1913, 60, 646.

Tests. (1) Ammonium sulphide gives a flesh-coloured precipitate which turns brown, and is soluble in dilute acids.

(2) A borax bead, dipped into a solution of a manganese salt and heated in the oxidising flame, becomes violet or lilac in colour, and loses its colour when heated in the reducing flame.

(3) Fused with sodium carbonate and a little potassium chlorate in a platinum capsule, manganese salts are oxidised to deep green manganate. The green mass dissolves in a *little* water to a green solution which becomes pink when greatly diluted with water or (better) when acidified and gently warmed.

(4) A solution of a manganese salt, made alkaline by addition of sodium hydroxide, gives a deep blue colour on addition of a drop of a 0·05 per cent. solution of benzidine in 10 per cent. acetic acid.

Group 3. GASEOUS POISONS

These poisons are grouped together for convenience in description, but do not really form a special group of pulmonary or respiratory poisons. There are four classes of poisons acting in the state of vapour.

1. The volatile acids, acting as irritants to the pulmonary mucous membrane, and setting up an intense bronchitis. These have already been considered.

2. Other powerful irritant gases, such as chlorine and fluorine, acting more by the irritation they produce than by their poisonous effects after absorption.

3. Gases which can be breathed, but after absorption act as definite poisons to the system, typically CO, AsH_3, various volatile substances, including those used as anæsthetics, and ordinary coal-gas. These are divisible into two sub-groups: (*a*) those which combine with the hæmoglobin to form fixed compounds; (*b*) those which apparently are dissolved in the plasma of the blood and poison the tissues to which they are distributed. To this group might be added the vapour of nickel carbonyl just considered.

4. Neutral gases, whose sole effect is due to the deprivation of the tissues of oxygen, *e.g.*, an atmosphere of nitrogen.

Owing to the fact that the poisonous material is in a state of high dispersion, and that in the air-cells of the lungs it meets with a large absorbing surface, and instantly enters the blood, the effects of gaseous poisons are rapid and powerful.

The majority of the poisonous gases are industrial products and are never likely to be met with in the ordinary atmosphere in sufficient quantity to produce injurious consequences except in war conditions. Hence fatal accidents, arising from their inhalation, most commonly occur in circumstances which can leave no question respecting the real cause of death. It is not necessary to consider all of these substances, but a knowledge of the properties of certain of the more common gases is essential to a medical jurist. Amongst these are carbonic acid, carbon monoxide, sulphuretted hydrogen and ordinary coal-gas. Agents of this description can rarely be employed with any certainty as instruments of murder, and if they were so employed the fact could be established only by circumstantial evidence. One alleged instance of murder by carbonic acid is, however, reported by Devergie, and there have been several

cases of murder and attempted murder by means of coal-gas. Death, when arising from the breathing of any of the gases, is generally, however, attributable to suicide or accident. In France it is by no means uncommon for a person to commit suicide by sleeping in a closed compartment, in which charcoal has been suffered to burn; in England there are many accidental deaths where coal or coke has been employed as fuel in small and ill-ventilated rooms, and coal-gas is the most common substance used for suicidal purposes. On such occasions a person may be found dead without, to the casual observer, any evident cause. The discovery of a body in these circumstances may create a suspicion of murder. In such a case it is obvious that the establishment of the innocence of an accused person may depend entirely on the discrimination and judgment of a medical practitioner. An instance, illustrative of the consequences of popular prejudice, occurred in London in 1823.

Six persons were lodging in the same apartment, where they were all in the habit of sleeping. One morning an alarm was given by one of them, a woman, who stated that on rising she found her companions dead. Four were discovered to be really dead, but the fifth, a married man, whose wife was one of the victims, was recovering. He was known to have been on intimate terms with the woman who gave the alarm, and it was immediately supposed that they had conspired together to destroy the whole party, in order to get rid of the wife. The woman, who was accused of the crime, was imprisoned, and an account of the supposed barbarous murder was soon printed and circulated in the metropolis. Many articles of food about the house were analysed, in order to discover whether they contained poison, when the circumstances were explained by the man stating that he had placed a pan of burning coals between the two beds before going to sleep, and that the doors and windows of the apartment were closed.[1] A set of cases of a similar kind, in which there was at first a strong suspicion of poisoning, has been reported.[2]

Poisoning by Carbon Dioxide or Carbonic Acid Gas (CO_2)

Sources and Methods of Occurrence. This gas is freely liberated in respiration, combustion, and fermentation. It is also produced in the calcination of chalk or limestone, and is sometimes diffused through the shafts and galleries of coal-mines, where it is commonly called "choke-damp." Carbonic acid gas is also met with in wells, sewers, cellars, and other excavations in the earth, in old brewers' vats, etc. In these cases it is generally found most abundantly close to the soil, or at the lower part of the well. The slow evaporation of water strongly charged with the gas, while trickling over the sides of these excavations, may further assist in contaminating the air. Damp sawdust or straw slowly absorbs oxygen from a confined atmosphere, and sets free carbonic acid.

Poisoning by this gas is, as might be anticipated from its sources, almost entirely a matter of accident, as in workmen when engaged in cleaning out vats, etc., from which many fatal cases are recorded.

In reference to suffocation by carbon dioxide, it is a matter of popular belief—and, in fact, it has been often asserted by writers on asphyxia—that the burning of a candle in a suspected mixture of carbon dioxide and air is satisfactory proof that the atmosphere may be breathed with safety. Observations have, however, tended to show that this statement is not to be relied on as affording an indication of security. A case is related by Christison where a servant, on entering a cellar in which

[1] Christison, p. 583.
[2] *Med. Gaz.*, vol. 36, p. 937.

grape juice was fermenting, was suddenly seized with giddiness. She dropped her candle on the floor, but had time to leave the cellar and shut the door behind her, when she fell down senseless. Those who went to her assistance found, on opening the door, that the candle was still burning. A candle will burn in air which contains even 10 or 12 per cent. of its volume of carbonic acid gas; and although such mixtures might not prove fatal to man, yet they would cause unpleasant symptoms such as giddiness and insensibility.

Degree of Toxicity and Fatal Dose. In reference to the fatal proportion of CO_2 in the air breathed, it is necessary to make a distinction between the contamination of air by the addition of a quantity of carbon dioxide, and the case where this gas is produced by combustion or respiration in a close apartment at the expense of the oxygen actually contained in the air. Every volume of carbon dioxide formed by combustion indicates at least an equal volume of oxygen removed. Such an atmosphere is, *cæteris paribus*, more destructive than another where the air and gas are in simple mixture. If we assume that in each case the noxious atmosphere contains 10 per cent. of carbon dioxide, then in one instance there will be nearly 7 per cent. more of oxygen and 7 per cent. less of nitrogen than in the other, since the production of 10 parts of carbon dioxide as a result of combustion implies the loss of 10 parts of oxygen. This difference in the proportions may not be, practically speaking, exact, because there is no apartment sufficiently closed to prevent air rushing in from the exterior while combustion is going on within it; but, nevertheless, the above statement may be taken as an approximation to the truth. When the gas is respired in its lowest poisonous proportion the symptoms come on more slowly, and the transition from life to death is frequently tranquil. This is what we learn from the histories of suicides. The symptoms in such cases appear to resemble closely those which indicate the progressive influence of opium or other narcotic poison on the body.

In a case of alleged murder by charcoal vapour in Paris, a question was put to a medical witnesses respecting the *quantity of charcoal* required to be burnt in a particular chamber in order to asphyxiate two adult persons. This question could only be answered approximately: because in burning charcoal, the sole product is not carbon dioxide, a variable quantity of carbon monoxide usually being evolved. Then, again, much of the carbon dioxide formed may escape in various ways from an imperfectly closed apartment. An attempt was made to infer the quantity of charcoal consumed from the weight of ashes found in the apartment, but no satisfactory answer could be given to this question. The prisoner was, however, convicted of murdering his wife by charcoal vapour.

With regard to the dosage of CO_2 (or any other gas for that matter) required for a fatal result, it is useless at a time remote from the accident to make any analysis of the general atmosphere of the place where the fatal event took place. This arises from the well-known laws of the diffusion of gases and the effects of heat upon this diffusion and their expansion. To be of the slightest value as evidence, such analyses must be made (1) from the actual vicinity of the body, and (2) at the moment of discovery, though even this is probably too late.

It is well known, by the effects of the vapour of a lime-kiln, that one person lying at the edge of the kiln may be destroyed, while another at ten yards' distance, either on the same or at a lower level, may entirely escape ; and it would not be possible in such a case to speculate upon the proportion of gas which had destroyed life, except by collecting the air from the spot where the accident occurred, and at or about the time of its occurrence. Another fallacy appears to be, that because a dead body is found recumbent, it is to be inferred that the person must have lain down and have been destroyed while sleeping. The dead body of a person must always be found thus lying on the floor, unless it be supported ; but suffocation may have actually taken place, or at least have commenced, when the deceased was in the sitting or erect posture.

Duration. The duration of symptoms of poisoning by carbon dioxide varies according to the concentration in which it is present in the atmosphere respired. Undiluted carbonic acid gas is respired with difficulty, or not at all, and produces spasm of the glottis, and almost instant death. With smaller percentages death is slower in onset, though unconsciousness may be very rapid.

Symptoms. In severe cases the symptoms commonly observed are as follows :—Sensations of great weight in the head, pressure in the temples, ringing in the ears, with a pungent sensation in the nose ; a strong tendency to sleep, accompanied by giddiness, and so great a loss of muscular power, that, if the person be at the time in an erect posture, he falls to the ground. The breathing, which is observed to be at first difficult and stertorous (snoring), becomes suspended. The action of the heart, which on the first accession of the symptoms is very violent, soon ceases : sensibility is lost, and the person now falls into a profound coma, or state of seeming death. The warmth of the body still continues ; the limbs are relaxed and flexible, but they have been observed in some instances to become rigid or even convulsed. The countenance is livid or of a leaden colour, especially about the eyelids and lips, but on some occasions it has been pale and placid. The access of these symptoms is stated to have been sometimes accompanied by a pleasing sensation of delirium, while at others the most acute pains have been suffered. In some instances there appears to have been irritability of the stomach, for the affected person has vomited the contents of his stomach in a semi-digested state. Those who have been resuscitated have felt pain in the head, or pain and soreness over the body for several days ; while, in a few severe cases, paralysis of the muscles of the face has supervened on recovery. For other symptoms, *vide* " Asphyxia," Vol. I.

Treatment. There is only one chance for a victim, and that is to remove him into fresh air and employ artificial respiration if necessary. If pure oxygen is obtainable, it may be tried.

Post-mortem Appearances. It has been said that the body of a person who has perished from the inhalation of carbon dioxide retains the animal heat, *cæteris paribus*, for a longer period than usual ; and hence cadaveric rigidity does not commonly manifest itself until after the lapse of many hours ; in a case to be afterwards related, the body was, however, found to have cooled considerably within the short space of two hours ; there is, therefore, no reason to believe that this mode of death affects the rate of cooling or the access of rigidity. In some instances it is said the face has

been found livid and swollen and the features distorted, but more generally it has been pale and placid, as if the persons had died without a struggle in the position in which their bodies were found. The skin is sometimes livid or presents patches of lividity, and the limbs are quite flaccid. The pupils are dilated. *Internally*, the venous system is filled with liquid blood of a dark colour. In death from carbon dioxide, as a result of combustion, the blood has sometimes had a light-red colour; this is due to the co-existence of carbon monoxide in the products of combustion. The veins of the lungs and brain are observed to be especially over-full. The tongue appears swollen, and the mucous membrane of the intestinal canal is often interspersed with dark ecchymosed patches. The following appearances were met with thirty hours after death in the bodies of two adults, a male and a female, who died from the accidental introduction of carbon dioxide into their bedroom from burning ashes. Externally—there was nothing unnatural, excepting a few slight discolorations on the back of the man. Internally—there was injection of the membranes and great vessels of the brain. Each lateral ventricle contained about half an ounce of clear serum, the lungs were gorged with dark blood, and the lining membrane of the air-tubes (bronchi) was slightly reddened. The left sides of the hearts were nearly empty; the right contained a quantity of dark half-coagulated blood. The stomachs were healthy. The bodies were found on the floor of the bedroom in positions of ease. The deceased persons had had the power to get out of bed, but were unable to escape from the chamber. It will be perceived from this description that there is nothing very characteristic in the appearances, and thus it is always easy to ascribe death to apoplexy or some other cause; but it should be remembered that carbon dioxide itself is a narcotic poison, inducing cerebral congestion and apoplexy.

Analysis. Sometimes a medical witness may be required to state the nature of the gaseous mixture in which a person may have died. He will have but little difficulty in determining whether carbon dioxide is or is not one of the deleterious agents in such a mixture. When it exists in a confined atmosphere, its presence may be identified, if previously collected in a proper vessel, by the following characters: 1. It extinguishes a taper if the proportion be above 12 or 15 per cent.; and, from the great density of the gas, the smoke of the extinguished taper may be commonly seen to float on its surface. 2. Lime-water, or a solution of lead acetate gives an immediate white precipitate when poured into a jar of the gas; and the precipitate thus formed may be collected by filtration, and proved to possess the well-known properties of carbonate of calcium or lead. Air containing only 1 per cent. of carbon dioxide affects lime-water: if it amounts to 2 per cent. a few cubic inches will suffice to show its presence by the lime-water test. The *proportion* in which carbon dioxide exists in a mixture may be determined by introducing into a measured quantity, in a graduated tube over mercury, a strong solution of potash. Absorption will after a time take place, and the decrease in volume of the gas will indicate the proportion of carbon dioxide present. When this gas exists in a confined spot, as in a well or cellar, it may be generally got rid of by placing within the stratum a pan containing slaked lime, loosely mixed into a paste with water; by exciting combustion at the mouth of the pit; or, what is better when available, by a jet of high-pressure steam. Lives are often

successively lost on these occasions in consequence of one person descending after another, in the expectation of at least being able to attach a rope to the body of his companion. The moment that the mouth comes within the level of the invisible stratum of gas all muscular power is lost, and the person commonly sinks lifeless. Air may be collected for the purpose of testing by lowering a bottle filled with fine dry sand, by means of a string attached to the neck, and guiding the bottle by another string attached to its base. When the bottle is within the stratum it should be turned with its mouth downwards; and when the sand has fallen out, it may be rapidly raised, with its mouth upwards, by pulling the string attached to the neck. The bottle should be immediately stoppered and the contents examined.

Cases. Cases of poisoning by pure CO_2 are very rare; in almost all the alleged cases CO or SH_2 or other poisonous gases were almost certainly the agents; probably the only genuine ones are those occurring in old brewers' vats, *e.g.*:—

In 1863 a boy mounted on a forty-barrel vat, and while looking through the manhole fell among some wet hops and speedily died from respiring the atmosphere of carbonic acid. Two men successively endeavoured to rescue the boy, but each died in the attempt. In the same year a man at Bromley descended into a large vat, having previously applied the candle test. He was heard to cry out, "There is gas here," and he instantly fell back dead: he had probably stirred up the contents after he had lowered the candle.[1] Many other cases of a similar kind are reported. In these circumstances the noxious agent is pure carbonic acid more or less mixed with air.

The following, although described as CO_2 poisoning, was probably caused by a mixture of gases, with the possible inclusion of ammonia:—

On May 6th, 1904, three men, on descending into the hold of a vessel which arrived at Leith Docks with a cargo of guano, were overcome by carbonic acid gas, which had generated from the guano. All died after every means had been adopted to restore them to consciousness. Police Sergeant Hill, who pluckily went to their assistance, also collapsed, but recovered.

In the following case there is a suspicion of poisoning by nitrous fumes rather than by carbon dioxide:—

An old woman occupied a room under one in which there was a quantity of nitric acid kept in store. Owing to some accident a carboy was broken; the acid ran through the ceiling into the room below, acting upon and corroding the coverings of the deceased's bed. As the room was filled with nitrous fumes, a chemist was consulted, and he advised that whiting should be freely used for the purpose of neutralising the acid. This advice was followed, and several persons, who were in the room witnessing the operation, felt oppressed, and were obliged to leave it: they were observed to stagger, as if intoxicated, on reaching the street. The room was then completely closed, and the whiting allowed to remain in contact with the acid. The deceased had suffered from diarrhœa for a few days previously, and was obliged to resort to the night-chair, which was in the room in which the accident had occurred. As she remained absent half an hour, some persons entered the apartment, and found her in the chair unable to move. She was taken into another room, and on a medical man being called to her, he found her sleepy, comatose, and her mind confused; there was great difficulty of breathing, and extreme lividity of the face and lips; the arms and legs were cold, and the pulse was full. In spite of efforts made to save her, she died in about an hour from the time at which she had entered the room. Those who found her in the

[1] *Lancet*, 1864, 2, p. 552.

apartment do not appear to have suffered. This was a case of slow poisoning by carbonic acid, for no carbon monoxide could have been evolved from the action of the acid on the chalk. Age, and debility from previous illness, may account for the unusual circumstance that the deceased did not recover on being removed to a pure atmosphere.

Poisoning by Carbon Monoxide (CO)

Sources and Methods of Occurrence. Whenever carbon is burnt either at a high temperature or in a limited supply of oxygen some carbon monoxide is formed. The sources of the gas are therefore extremely numerous.

In the household the main source of the gas and the most common cause of poisoning is coal-gas, which normally contains 4 to 10 per cent. of carbon monoxide.

Coal-gas is the product of the distillation of coal, and its composition varies considerably. An analysis of coal-gas, as supplied in London, showed that it contained per cent. : Hydrogen, 46·43 ; marsh gas, 38·9 ; carbon monoxide, 5·62 ; oleficient gas, 3·86 ; water vapour, 2·48 ; nitrogen, 2·22 ; carbon dioxide, 0·46. Carbon monoxide is the chief poisonous substance in coal gas, but there is little doubt that the heavier hydrocarbons also have a noxious influence. Coal-gas owes its peculiar odour chiefly to the vapour of naphtha, which thus indicates its presence. Suicide by means of coal-gas is, of course, very prevalent.

Water-gas is made by passing steam over hot coke, and it is essentially a mixture of hydrogen and carbon monoxide, containing 45·50 per cent. of the latter. It is commonly mixed with coal-gas for use as an illuminant, and on account of its high carbon monoxide content, it is extremely dangerous.

Other common household sources are bath geysers, other types of water-heaters and slow-combustion stoves. Charcoal fires and braziers are not often found in households, but they are often the cause of accidental death in night watchmen who take a brazier into their cabin to keep themselves warm. In these cases, the poisonous effects are probably the result of both carbon monoxide and carbon dioxide, and the same is true of poisoning by the vapours of lime kilns, etc.

Less obvious sources of the gas are found in defective vents from a room below causing a leak of gas into a room on the flat above and percolation of the gas from a broken main in the street into houses adjoining.

In industry, blast furnace gases are rich in carbon monoxide, and acute and chronic poisoning may occur in the workers.

Blasting operations in mines lead to a considerable output of CO, while explosions from coal-dust lead to serious cases of poisoning from the same gas (after-damp). Gas may accumulate in old mine workings from seepage.

Exhaust fumes from motor cars and other petrol-burning engines contain varying amounts of carbon monoxide according to the relative efficiency of the carburation employed. On an average these gases contain about 6 per cent. of carbon monoxide, and therefore an engine running in a closed garage will render the air unfit to breathe in a very short time. Yandell Henderson considers that at least 1 cubic foot per minute is given out for each twenty horse power. Apart from small private garages, where accidents from this source are all too common,

workers in assembling and repair shops where exhaust gases are not efficiently removed are liable to suffer. It is also possible, owing to leaking exhaust pipes, favourable wind, etc., for exhaust gases to gain access to the bodies of closed cars, aeroplanes, motor-boats, etc., and so cause symptoms of poisoning in the passengers or driver.

Apart from the motor industry, workers in laundries, pressers in tailors' shops, metal moulders, linotypists, furnace tenders, engine-room workers in motor-driven ships, etc., may suffer unpleasant or even dangerous symptoms from absorption of the gas.

During the process of burning lime there is an evolution of CO_2 and CO, and a certain number of deaths occur among tramps who sleep beside these kilns for warmth in the winter.

In warfare the evolution of carbon monoxide from the bursting of high explosives, from the operation of machine-guns in bomb-proof shelters, from the use of explosives in sapping, etc., causes a considerable number of accidents.

Carbon monoxide in ethylene: Sherman and others[1] report three cases of collapse under ethylene anæsthesia, of which two were fatal. The blood of the patients showed 50 to 60 per cent. saturation with CO. The gas cylinder was found to contain 0·7 per cent. carbon monoxide.

Nature and Toxicity of the Gas: Mode of Action. Carbon monoxide is a colourless odourless gas, which is devoid of irritating effect on the respiratory passages, and therefore gives no warning of its presence. It unites readily with hæmoglobin forming a compound which is more stable than oxyhæmoglobin, and therefore readily displaces oxygen from the latter compound. The power of carbon monoxide to combine with hæmoglobin is between 200 and 250 times as great as that of oxygen. This means that if an atmosphere is breathed containing only 0·1 per cent. of carbon monoxide (*i.e.*, $\frac{1}{200}$, approximately, of the amount of oxygen), rather more than half the hæmoglobin of the blood will ultimately become combined with carbon monoxide, and so be rendered useless for conveying oxygen to the tissues.

Hill[2] and Barcroft[3] have shown that it combines more readily with unsaturated oxyhæmoglobin than with oxygen-free hæmoglobin.

Although carbon monoxide hæmoglobin is more stable than oxyhæmoglobin, it is by no means a firm compound, and dissociation readily occurs when breathing takes place in an atmosphere of oxygen or ordinary air. Probably the whole of the gas disappears within a few hours. The red cells do not appear to be damaged in any way by saturation with the gas, but readily resume their function as oxygen-carriers. Carbon monoxide is not toxic in itself, and it does not form a toxic compound with the blood or tissues. It acts as a poison entirely by virtue of the fact that it deprives the tissues of oxygen. This applies to carbon monoxide as a separate gas, but it may not be correct with regard to coal-gas and other mixtures of gases, which may exercise specific toxic effects on protoplasm.

The effects of carbon monoxide therefore are to be considered as due to oxygen starvation, and the symptoms are those of anoxæmia. If, however, the oxygen is cut off for a sufficiently long time, serious damage

[1] *Jour. Amer. Med. Assoc.*, 1926, 86, 1765.
[2] *Biochem. Jour.*, 1914, 7, 471.
[3] *Ibid.*, 481.

to cellular tissues may result. In prolonged coma from CO poisoning, therefore, there may be permanent damage to highly developed cells, such as those of the brain. Lesions of the heart and severe congestion of the lungs may occur from the same cause, and probably more or less broncho-pneumonia is the rule in severe cases of gassing.

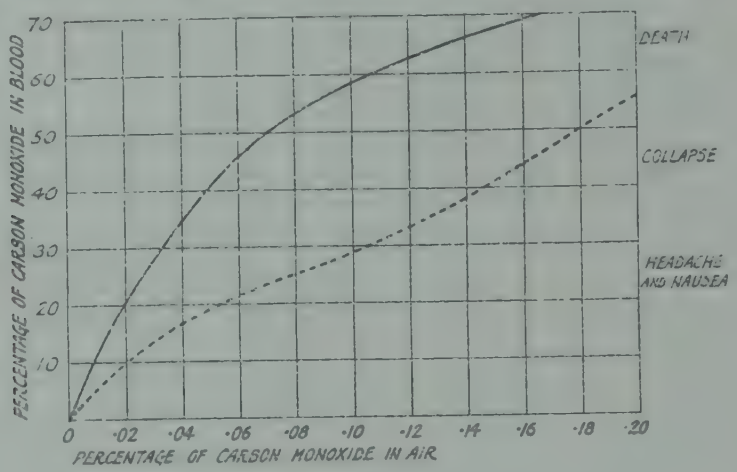

FIG. 3.

Hill and Semerak[1] state that CO poisoning causes bilateral necrosis of the lenticular nucleus, thus bearing out an earlier observation of Kolisko. They also found punctiform hæmorrhages in the white matter of the brain and more or less œdema.

Haggard[2] has shown that pure CO has no toxic action on neuroblasts, but that coal-gas has such an effect. It may be accepted from the experimental work of Haldane in this country, and of Yandell Henderson and Haggard in the United States, that carbon monoxide *per se* has no toxic effect.

Haldane has shown that a person sitting at his ease uses less than half the oxygen supplied by the blood, and therefore the blood may become seriously charged with carbon monoxide before symptoms manifest themselves, although even in this state rapid exertion may cause collapse. If the blood becomes more than half saturated he may collapse even if he remains at rest.

Henderson and Haggard investigated the rate at which carbon monoxide is absorbed, and the physiological effects produced by exposure to varying concentrations for one hour. The results of these investigations are shown in the graph,[3] Fig. 3.

"The upper curve is the theoretical dissociation curve of carbon monoxide and hæmoglobin in the presence of air: the lower curve shows the approximate percentage of saturation that a man will develop in one hour, if he is sitting still in an atmosphere containing the amount of carbon monoxide shown in the scale below the abscissa. This curve and the scale are drawn for a normal volume of breathing. If respiration were doubled, these percentages of saturation would be attained in half an hour, and the curve would therefore represent the percentages of saturation attained in this time by a man breathing air containing the amounts of carbon monoxide indicated. . . .

[1] *Jour. Amer. Med. Assoc.*, 1918, 81, 644.
[2] *Amer. Jour. Physiol.*, 1922, 60, 244.
[3] *B.M.J.*, January 9th, 1926, p. 44.

"When the time of exposure is measured in hours and the concentration is measured in parts of carbon monoxide per 10,000 of air, then, if the product of the time multiplied by the concentration equals 3, no perceptible physiological effect results. If the product equals 6, there is a just perceptible effect, perhaps a slight headache and lassitude; if it equals 9, severe headache and nausea result; if it equals 15, the condition is dangerous: if it rises above 15, the conditions are such as will be quickly fatal. If the subject is making any muscular exertion, and his breathing is increased, the figures for the products of time and concentration that are comfortable and safe are reduced from 3 to 2, or 1, or even less, for the rate of absorption varies directly with the volume of the breathing."

Henderson estimates that death will occur if an atmosphere containing 2 parts per 1,000 of CO is breathed for four hours or 4 parts per 1,000 for one hour.

Vale,[1] from experiments on himself, concludes that where no work is undertaken in atmospheres containing CO up to 1·14 per 1,000, 50 per cent. of the amount inhaled is the maximum that will be absorbed. When work is being done, the respirations are increased, and the amount absorbed may be twice to 4 times as much as when at rest, and even with 1·14 per 1,000 consciousness may be lost on exertion. With 2 parts per 1,000 muscular power is completely lost, and the victim becomes rapidly unconscious; while with concentrations of 3 parts per 1,000 death is almost inevitable unless rescue is effected very promptly.

These figures are, of course, only approximate, and both age and size may affect the toxicity of carbon monoxide. Leschke records a case indicating the greater sensitivity of children, and one may recall the use of mice and birds as "indicators" of carbon monoxide in mines, etc.

Symptoms. The rapidity of onset and the nature of the symptoms depend on the concentration of the gas in the blood, and therefore on its concentration in the air.

In high concentrations a few breaths are sufficient to saturate the blood and unconsciousness may supervene with startling suddenness. With lower concentrations little effect is felt until the blood is saturated to about 20 per cent., when headache and lassitude are observed. When it reaches about 30 per cent. there is nausea, giddiness, throbbing in the head, ringing in the ears, dyspnœa, and muscular weakness. The muscular weakness, inco-ordination and giddiness increase rapidly with increasing saturation up to 50 per cent., while between 50 and 60 per cent. paralysis and coma supervene.

The respiration is usually rapid and stertorous, the pulse-rate increased, and the blood-pressure may be reduced. In some cases there may be a stage of violent delirium. Reflexes and sphincter control are lost. Vomiting may occur. The respirations and pulse weaken, and the patient ceases to breathe.

One of the first effects observed is loss of strength. Even in a person awake and active the gas may speedily produce inability to move or to call for assistance. Sir Thomas Stevenson had personal experience of this. In one instance a charcoal brazier was left, only for a short time, in the cell of a prison. It was removed, and the prisoners went to sleep. They

[1] *Trans. Inst. Mining Engin.*, 1922, 73, 417.

experienced no particular effects at first, but after some hours two were found dead. Thus, then, an atmosphere which can be breathed for a short time with impunity may ultimately cause death.

For a very graphic account of the symptoms, *vide B.M.J.*, 1898, 2, p. 33. Dr. Le Neve Foster reports as follows :—

"The poison took effect most suddenly; probably its action was accelerated by the exertion of climbing rapidly. I felt decidedly queer when I reached the 115-fathom level, and thought a drop of brandy might revive me. I took out my little brandy flask, but already my fingers seemed incapable of doing the work properly, and someone unscrewed the stopper for me. I took a small sip and sat down. Everything then seemed in a whirl, and the atmosphere seemed to be a dense white fog. This must have been, so far as I can judge, a little before 1 p.m. . . . Sitting next to me was Mr. Williams, and within a few feet were Captain Reddicliffe and Henry Clague; the men who had remained all the time at the 115-fathom level, or at all events had not descended as low as we did, had started to climb to the surface, but of their starting I have no recollection. A curious fact is that we all sat without moving or trying to escape; the foot of the ladder was close by, yet none of us made any effort to go to it and ascend even a single rung. We none of us tried to walk a dozen steps, which would have led us to the other side of the shaft partition, where we all knew that there was a current of better air. We simply sat on and on."

Treatment. As the length of time during which the anoxæmia is present is more important than the extent of saturation, especially when patients are insensible, treatment must be directed towards the most rapid resumption of normal breathing.

If the anoxæmia continues for a long time, irreparable damage is likely to occur in the cells of the cerebral cortex and elsewhere.

The first step is to remove the victim into fresh air. If he is conscious and breathing easily, no treatment is required, as he will recover if left to himself. In these cases no permanent damage is likely to occur, and elimination of the gas takes place rapidly, about half the quantity being lost in the first hour.

With higher degrees of gassing artificial respiration must be started forthwith. Henderson has shown that in extensive gassing there is not sufficient carbon dioxide in the blood to cause the necessary stimulus to the respiratory centre. The patient therefore may be in an atmosphere of pure oxygen, and though the lungs are full of this gas, natural breathing will not take place. He has suggested and has proved the value of adding 5 per cent. of carbon dioxide to the oxygen which is used. This stimulates the respiratory centre, considerably improves the ventilation in the lungs, and thus causes a rapid removal of the carbon monoxide.

If the mixture is not available, an attempt may be made to cause the patient to breathe in the expired air of the medical attendant. This can be carried out by making a tube of stiff paper through which the attendant may pass his breath at the moment when the patient is making an inspiration. Warmth should be applied to the extremities and the body well wrapped up. The tendency to congestion of the lungs and possibly to pneumonia should be remembered.

Post-mortem Appearances. The surface of the body and the areas of hypostasis may present a bright cherry-red colour. This may also be seen about the face and lips, but is not always observed in this locality.

On opening the body the blood and the whole of the internal organs appear pink in colour, and this is especially seen in the walls of the intestines. The lungs on section show a bright red colour, and if the gassing has been extensive, they may appear solidified in parts. Microscopic sections show œdema and hæmorrhages in many cases. The brain is commonly engorged, and more or less œdema is usually found. There may be minute punctiform hæmorrhages in the cortex and basal ganglia.

Ischæmic necrosis of the lenticular nucleus may be found. These changes are the result of anoxæmia, and explain the reason of impaired cerebral function which occasionally arises after carbon monoxide poisoning.

Gürich[1] has described lesions of the heart in three cases of poisoning from coal-gas. He found acute myocarditis and punctiform hæmorrhages in the muscle. He also found broncho-pneumonia with hæmorrhages in the lower lobes of the lungs, and softening of the globus pallidus of the brain. He points out that the lesions of coal-gas poisoning are not necessarily those of carbon monoxide poisoning alone, and in this he is undoubtedly justified.

Analysis. With regard to the value of chemical tests for carbon monoxide, the reader is referred to the remarks on pp. 472-3 which refer equally to air contaminated with CO, or indeed, any other gas. It must be remembered also, that the combination of hæmoglobin and carbon monoxide is loose, and that carbon monoxide hæmoglobin gradually decomposes when exposed to pure air, the carbon monoxide being lost. It follows that in the living subject, blood for analysis must be obtained as soon as possible after the exposure to the poisonous atmosphere, and that the drawn blood must be submitted to analysis as quickly as possible and with the minimum of exposure to air. Even after the severest poisoning, CO will have disappeared completely from the blood within twenty-four hours, and most of it within an hour of removal from the noxious atmosphere. In the cadaver, the disappearance of CO is much slower owing to the cessation of respiration and circulation. Martin (*Ann. Méd. Légale*, 1921, 1, 20) states that he has detected CO in the putrid exudations of cadavers three months after death. Once the blood has been drawn and is exposed to air, however, loss of CO again begins, so that here also speed is essential in carrying out the analysis.

The presence of considerable amounts of carbon monoxide in blood or blood-tinged fluids may be demonstrated very simply by the persistent red or pink colour. Thus on dilution with 100 volumes of water, blood rich in CO-hæmoglobin remains pink, while normal blood appears yellow. Similarly, blood containing much carbon monoxide remains red after treatment with reducing agents (*e.g.*, stannous chloride, ammonium sulphide, ferrous sulphate, etc.), while normal blood becomes brown or black. These tests, however, are very crude, and for confirmatory purposes, as well as for the detection of smaller concentrations of carbon monoxide, more delicate methods are required. The following procedures are adapted either for qualitative detection of carbon monoxide in blood, or for its quantitative determination.

1. *Tannic Acid.* For the qualitative test, equal volumes (5 c.c. is a convenient amount) of the sample are introduced into large stoppered

[1] *Munch. Med. Woch.*, 1925, 72, 2194.

test-tubes or small flasks, to each of which is added 1 c.c. of a 10 per cent. potassium ferricyanide solution. After the two flasks have stood undisturbed for ten to fifteen minutes, one of them is shaken for a further ten minutes, the stopper being occasionally removed to admit fresh air. This procedure removes any carbon monoxide which may be present. Both flasks are then treated with a few drops of yellow ammonium sulphide and 10 c.c. of a 10 per cent. solution of tannic acid. The blood in the shaken flask (containing no carbon monoxide) gives a dirty olive-green precipitate; the unshaken blood gives a precipitate varying in colour from the same dirty green to bright red according to the amount of carbon monoxide it contains. A distinct difference in colour is given by blood in which 5 per cent. of the hæmoglobin is combined with carbon monoxide.

Henderson and Haggard[1] have adapted this test to the quantitative determination of carbon monoxide in blood. They prepare a series of standard mixtures as follows:

C.c. blood saturated with CO, 0·0, 0·1, 0·2 . . . 0·9, 1·0;
C.c. normal, untreated blood, 1·0, 0·9, 0·8 . . . 0·1, 0·0.

These represent bloods in which, 0, 10, 20 . . . 100 per cent. of the hæmoglobin is combined with carbon monoxide, and should be freshly prepared. From the sample to be tested, and each of these standard mixtures, 0·1 c.c. is measured, and mixed with 0·4 c.c. of dilute ammonia (1 c.c. of 0·880 ammonia in 1,000 c.c.) without shaking. To each tube, 1·5 c.c. of a freshly prepared 1 per cent. tannic acid solution is then added. The whole series of tubes is then stoppered and allowed to stand overnight, after which the colour of the test sample is compared with the standard colours. It has been claimed that the colours are sufficiently stable to justify the preparation of standard tubes for permanent use, but it seems safer to prepare fresh standards as required.

Sayers and Yant[2] employ a similar method, preparing a series of standard mixtures of COHb and HbO_2 as above. They dilute the untreated and CO-saturated bloods to ten times their original volumes before making up the mixtures, and use 1 c.c. of each mixture. To each tube they add 1 c.c. of a solution made by mixing equal volumes of 2 per cent. tannic acid solution and 2 per cent. pyrogallol solution. The standard solutions are then sealed by pouring melted paraffin wax into the tubes, and in this state they keep for about two weeks.

Of the blood sample to be tested, 0·1 c.c. is diluted to 2·0 c.c. with water, and about 40 mgrm. of a mixture of tannic acid and pyrogallol in equal proportions is added at once. The colour develops in thirty minutes, when comparison is made with the series of standard tubes.

2. *Spectroscope.* The absorption spectrum of carbon monoxide hæmoglobin is very similar to that of oxyhæmoglobin, the most obvious bands being two between the D and E lines. The first band, near the D line, very nearly coincides with the corresponding oxyhæmoglobin band (the mid points are at wave-lengths 571 and 578 respectively); the second is also very close to its oxyhæmoglobin analogue, but a little nearer the violet end of the spectrum. In both cases, however, the difference between the CO-hæmoglobin band and the oxyhæmoglobin

[1] *J. Amer. Med. Assoc.*, 1922, 79, 1143.
[2] U.S. Bureau of Mines, 1925, Technical Paper No. 373; *Analyst*, 1926, 51, 99.

band is too slight for a mixed spectrum to give more than two bands – *i.e.*, there is always overlapping. Yellow ammonium sulphide, however, reduces oxyhæmoglobin to hæmoglobin, whose absorption spectrum consists mainly of a broad diffuse band extending from the D line almost to the E line. This reagent has no effect on CO-hæmoglobin. Consequently, if blood containing carbon monoxide is diluted, and treated with a few drops of yellow ammonium sulphide, the two-band spectrum persists. The test is not very delicate, and it is usually possible to see the two bands of CO-hæmoglobin superimposed on the single band of reduced hæmoglobin only if at least 25 per cent. of the total hæmoglobin is combined with carbon monoxide.

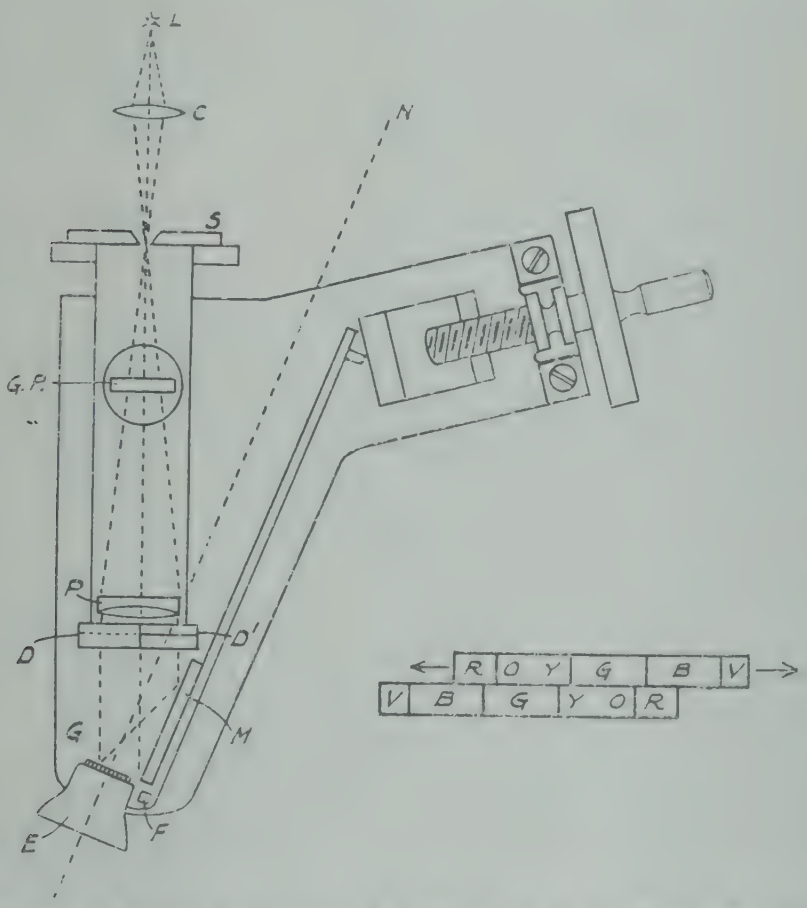

FIG. 4.—Diagram of Reversion Spectroscope. L = light source. C = condenser. S = adjustable slit. G.P. = glass-plate micrometer. P = achromatic lens. D and D' = weak prisms, base up and base down respectively. G = grating. F = fulcrum. M = stainless steel mirror. E = eye-piece. N = normal to grating. The spectra are seen side by side, one reversed in direction to the other; the upper one moves horizontally on turning the micrometer screw.

3. *Hartridge Reversion Spectroscope.*[1] The reversion spectroscope devised by Professor Hartridge renders the spectroscopic detection of carbon monoxide much more delicate, and also allows of approximately quantitative determination. The instrument produces two spectra, side by side, and identical except that they are reversed in direction (Fig. 4). Arrangement is made for moving one of the spectra laterally and for measuring the amount of movement. To calibrate the instrument, a rectangular glass trough, divided by a diagonal glass partition is used. One compartment is filled with normal blood diluted to fifty

[1] *Proc. Roy. Soc.*, 1923, A, 102, 575.

times its volume with distilled water; the other is filled with another sample of the same blood, saturated with carbon monoxide, and similarly diluted. Then, as Fig. 5 shows, when the trough is placed in front of the spectroscope, the spectrum seen will be that of a mixture of oxy- and carbon monoxide-hæmoglobin in the ratio of the thicknesses of the two compartments along the line joining the slit and the light source. The trough is first adjusted so that the spectrum of oxyhæmoglobin only

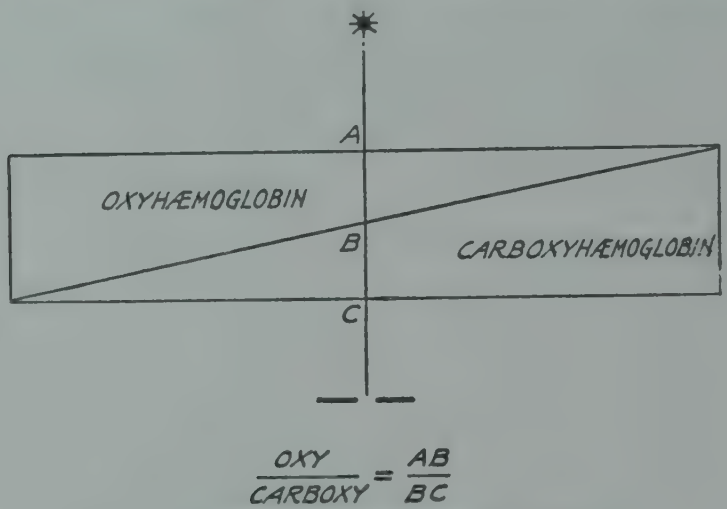

FIG. 5.——Rectangular trough divided diagonally.

is seen and the micrometer screw is turned until the two bands nearest the D line coincide (Fig. 6A). The micrometer reading is that for absence of CO-hæmoglobin. The trough is then moved until only carbon monoxide hæmoglobin is seen, and the spectra, which have moved apart as in Fig. 6B, are brought into coincidence again (as in Fig. 6A) by movement of the micrometer screw. The micrometer reading now corresponds to the presence of 100 per cent. carbon monoxide hæmoglobin. A series of intermediate readings enables a graph to be constructed relating the reading of the micrometer screw to the percentage of hæmoglobin in combination with carbon monoxide. Thereafter it is only necessary to view the double spectrum of a suspected blood sample (suitably diluted), bring the two narrow bands near the D line into alignment, and compare the micrometer reading with the graph. It is claimed that this method can detect the combination of 1 per cent. of the hæmoglobin with carbon monoxide.

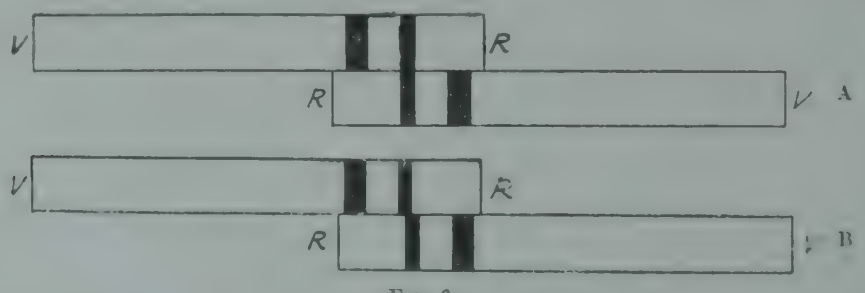

FIG. 6.

4. *Gasometric Method.* Van Slyke and Neill[1] have described a method for estimating the CO content of blood by means of their manometric apparatus. The method is claimed to give results accurate to 0·2 vols.

[1] *J. Biol. Chem.* 1924, 61, 523.

per cent., using 2 c.c. of blood, and therefore to be satisfactory in cases of carbon monoxide poisoning. The blood is mixed in the apparatus, with an acid solution of potassium ferricyanide and saponin. The mixture is submitted to a Torricellian vacuum, in which the blood gases, CO_2, O_2 and CO (if present) are expelled. The CO_2 and O_2 are then absorbed by addition of an alkaline solution of sodium hyposulphite, and the residual carbon monoxide is measured manometrically after adjustment to a convenient volume—2·0 c.c. or 0·5 c.c. A correction must be applied for the small amount of nitrogen which is present, and for the amount of CO reabsorbed during the manipulation. The reagents, of course, must be free from dissolved gases.

A more accurate method has been devised by Sendroy and Liu.[1]

Full details of these methods are given in "Quantitative Clinical Chemistry, Vol. II.—Methods," by Peters and van Slyke.

Roughton and Root[2] have recently described a manometric method applicable to small volumes of blood (0·5 to 2·0 c.c.) which is particularly suitable for the estimation of small concentrations of carbon monoxide (2–5 vols. per cent.), such as may be encountered in cases of chronic poisoning.

5. *Palladium chloride.* Carbon monoxide reduces palladium chloride to metallic palladium, and various methods of estimation have been based on this reaction.

The gas, liberated from the blood by aspiration with a current of pure air, is passed first through lead acetate (to remove H_2S) then through dilute sulphuric acid, and finally through a standard solution of pure palladous chloride. The air is purified before reaching the blood by passage through palladous chloride. The liberation of gas from the blood is aided by addition of sodium hydroxide or potassium ferrocyanide. Quantitative results are obtained by estimation either of the reduced palladium (*e.g.*, Fodor's method[3]) or of the excess of palladium chloride (*e.g.*, Christman and Randall[4]). Fodor filters off the palladium, reconverts this to the chloride, and titrates with potassium iodide: Christman and Randall employ a colorimetric method.

Gray and Sandiford[5] have evolved a simple method, based on the absorption of carbon monoxide by palladium chloride, applicable to 0·5 c.c. of blood, but accurate only to within about 1 vol. per cent., at CO concentrations over about 5 vols. per cent. The carbon monoxide is absorbed by palladous chloride from diluted acidified blood in a Conway micro-diffusion apparatus, and the excess palladium chloride (after removal of the precipitated palladium) is determined colorimetrically by means of the red colour developed on addition of gum ghatli and potassium iodide.

The determination or detection of carbon monoxide in air can be carried out (1) by passing a considerable volume of the air through a solution of palladium chloride (the presence of H_2S, $(NH_4)_2S$, O_3 and H_2, must be excluded), (2) by passing the air through an acid solution of cuprous chloride which absorbs carbon monoxide (the absorbed gas is

[1] *J. Biol. Chem.* 1930, 89, 133.
[2] *J. Biol. Chem.* 1945, *160*, 123. Cf. also *J. Biol. Chem.*, 1941, *137*, 617.
[3] Quoted by Peterson, Haines and Webster, "Legal Medicine and Toxicology," 1923, vol. II, 321.
[4] *J. Biol. Chem.*, 1933, 102, 595.
[5] *Analyst.*, 1946, *71*, 107.

expelled by boiling and passed into palladium chloride solution where it causes precipitation of palladium), (3) by shaking the air with blood and testing the blood for the presence of carbon monoxide hæmoglobin by one or other of the several available methods. Ordinarily, for accurate quantitative work it is necessary to take into account the fact that in presence of oxygen the absorption of CO is incomplete, and, using human blood, the percentage of CO in the air is given by the equation :

$$\text{per cent. CO} = \frac{\text{per cent. of total Hb combined with CO}}{100 - \text{per cent. of total Hb combined with CO}} \times \frac{20{\cdot}93.}{210}$$

If, however, both the air sample and the blood are freed from oxygen, and sufficient blood is used, CO is absorbed to the extent of 94 per cent. even when it forms only 0·05 per cent. of the sample.

An accurate method suitable for carbon monoxide concentrations in air from below 0·05 per cent. to about 0·7 per cent. is given by Roughton and Root[1]. It is based on the absorption of the carbon monoxide by diluted blood under controlled conditions and the manometric determination of the CO-hæmoglobin produced.

A simpler, though less accurate method capable, however, of detecting 0·01 per cent. of carbon monoxide in ten litres of air, is fully described in Leaflet No. 7 of the series, on Methods for the Detection of Toxic Gases in Industry (Dept. of Scientific and Industrial Research), published by H.M. Stationery Office. The method is based on the reduction of palladium chloride to palladium. The air, at room temperature, is freed of interfering gases by being drawn through activated charcoal (a very convenient form of apparatus is described) and is then drawn through test paper freshly impregnated with the reagent (0·1 g. palladium chloride dissolved in 20 ml. distilled water and 20 ml. acetone). The grey-brown deposit of palladium is compared with standards (provided with the leaflet).

Banham, Haldane and Savage[2] have described a case in which the *post-mortem* signs indicated carbon monoxide poisoning, but in which they considered the colour changes to be due to the formation of **nitric oxide hæmoglobin** after death. The blood remained pink on dilution, and on adding a drop of sodium hydroxide gave a pink precipitate with tannin, and gave a two-banded spectrum similar to oxyhæmoglobin and carboxyhæmoglobin. These are the usual tests for CO–Hb.

On boiling a solution of the blood containing NO–Hb a pink coagulum was obtained. Nitrites given before death do not produce this compound, but change hæmoglobin to methæmoglobin. On addition of further nitric oxide this is reduced and converted into NO hæmoglobin. The writers suggest that nitrifying organisms may have obtained access to the blood and that the change may have occurred *post-mortem*. Banham suggests that certain cases of pneumonia which he saw in France in which the blood was distinctly pink may have been infected with a nitrifying organism of this nature.

We have frequently observed this colour in the lungs in cases dying suddenly from acute pneumonic influenza, but have never seen a generalised pink colour such as is found in carbon monoxide poisoning. The differential diagnosis is readily made spectroscopically, as the bands of

[1] *J. Biol. Chem.*, 1945, *160*, 135.
[2] *B.M.J.*, August 1st, 1925.

NO–Hb are displaced towards the red end of the spectrum relatively to CO–Hb. This is readily detected with Hartridge's reversion spectroscope.

Chronic Poisoning. The constant inhalation of small quantities of carbon monoxide produces a series of symptoms which are by no means serious in the majority of cases, but which may lead to continued ill health. Symptoms consist of headache, lassitude, breathlessness on exertion and a certain degree of muscular weakness, the general condition resembling neurasthenia. The patient not infrequently exhibits giddiness on looking upwards, and in long standing cases there may be unsteadiness in gait or ataxia.

The condition may be found in many workmen in the trades mentioned above. In addition to these trade risks, information is accumulating to show that there is a definite street risk of slight gassing with CO in many cities owing to the extensive use of motor transport in narrow streets. According to Wilson,[1] patrol men in congested districts of Philadelphia have complained at the end of the day's work of headache, nausea and weakness. In the blood of some of these about 30 per cent. of the hæmoglobin was found saturated with CO. This state of affairs promises to be worse unless steps are taken to improve the combustion of petrol in motor engines and fix a standard for exhaust gases or to discharge the exhaust upwards by means of a vertical pipe, as Henderson has suggested.

After-effects of Gassing. Many after effects of gassing have been recorded, the principal effects being a dulled mentality and loss of memory, pneumonia, heart lesions, paralysis and mental disease. Headaches, sometimes of a violent character, may occur and occasionally neuritis and sensory disorders may be observed. Fortunately these sequelæ are extremely rare.

Relapsing cases have also been described in which serious symptoms have occurred after a period of recovery.[2]

Poisoning by Sulphuretted Hydrogen (Hydrogen Sulphide) (SH_2)

Source and Method of Occurrence. Sulphuretted hydrogen gas is largely used or produced in connection with many chemical industries, *e.g.*, in the nickel and cobalt industry, in de-arsenicating acids, in sulphur dye works, and in the manufacture of leather. It is produced in the manufacture of artificial silk by the viscose process, in the soda industry, in the preparation of carbon bisulphide, in the manufacture of illuminating gas, in blast furnaces, etc. It is also generated in large quantities in putrefactive processes, especially in sewers and cesspools.

The men who were engaged in working at the Thames Tunnel suffered severely, during the excavation, from the presence of this gas in the atmosphere in which they were obliged to work. The air, as well as the water which trickled through the roof, was found to contain sulphuretted hydrogen: it was probably derived from the action of the water on iron-pyrites in the clay: it issued in sudden jets. Poisoning by this gas is always either accidental or (very rarely) suicidal. The very offensive odour which a small portion of it communicates to a large quantity of air is sufficient to announce its presence, and thus, with due caution, to prevent any dangerous consequences. The *Lancet*, 1903, I,

[1] *Jour. Amer. Med. Assoc.*, 1926, 87, 319.
[2] McConnel and Spiller, *Jour. Amer. Med. Assoc.*, 1912, 59, 2123.

p. 225, contains an interesting article on the subject by Thomas Oliver ; three fatal cases are there reported, with experimental observations on the cause of death.

It is well known that sulphuretted hydrogen in poisonous proportions may easily be given off from the very concentrated sewage contained in cesspools. It is often assumed, however, that the same danger cannot arise from the comparatively dilute liquid contained in ordinary sewers unless acid has been allowed to get into them and to come into contact with deposits containing sulphides, as in the case of the Lambeth accident. Dr. Haldane (*loc. cit.*) says :—

"The sewage ordinarily met with in well-built sewers does not contain sulphuretted hydrogen, nor does ordinary sewer air blacken lead paper. It seems probable that so long as sewage is sufficiently aerated there is no evolution of sulphuretted hydrogen, the bacteria simply oxidising the sulphur of the albumen to sulphates, and the carbon and hydrogen to carbonic acid and water, in the same manner as do the tissues themselves in the case of a living animal."

The East Ham case (below) is important as showing that in favourable conditions in summer the presence of acids or of sewage as concentrated as that of a cesspool is not essential.

In fatal cases Oliver found that methæmoglobin was always present, but he hesitated to ascribe death to this fact.[1]

Workers in chemical factories are quite familiar with sulphuretted hydrogen and its usual effects on the system ; for it is not by any means unusual for persons exposed to the fumes to become gassed—that is, they pass into a condition of insensibility lasting for different periods, and when they come round they are very often sick and dazed, and have a sense of oppression about the chest : and there is often a good deal of prostration for a day or two. Sometimes, the insensibility ends in death. It is, however, unusual for permanent effects to be produced upon the nervous system.[2]

Toxicity. The symptoms appear very rapidly when the gas is present in anything like large quantities, and may prove fatal within an hour (*vide* cases). In cases where the gas is more dilute the symptoms occur much more slowly. An atmosphere containing sulphuretted hydrogen, which may be breathed for a short time with impunity, may ultimately cause death.

Haggard[3] states that symptoms of irritation appear after a few hours' exposure to 0·01–0·015 per cent. of the gas. With 0·02–0·03 per cent. symptoms occur in an hour. Death may take place within half an hour by inhalation of 0·15 per cent., and immediate death by inhalation of 0·18 per cent. It is non-cumulative, and is readily oxidised.

Symptoms. The symptoms produced by sulphuretted hydrogen vary according to the degree of concentration in which it is breathed. When the air is but slightly contaminated with the gas, it may be breathed for a long time without producing any serious symptoms : sometimes there is a feeling of nausea or sickness, accompanied by pain in the head, or diffuse pains in the abdomen. When the gas is inhaled in a moderately diluted state, the victim soon falls senseless. According to the account given by

[1] *Lancet*, 1903, 1, p. 225.
[2] Wiglesworth, *B.M.J.*, 1892, 2, p. 124.
[3] *Jour. Ind. Hyg.*, 1925, 7, 113.

those who have recovered, this state of insensibility is preceded by a sense of weight in the stomach and in the temples, headache, giddiness, nausea, sudden weakness, and loss of motion and sensation. If the gas in a still less concentrated state be inhaled for some time, insensibility, coma, or tetanus with delirium supervenes, preceded by convulsions, or pain and weakness over the whole body. The skin in such cases is usually cold; the pulse irregular, and the breathing laborious. The symptoms are often observed to affect those who are engaged in chemical manipulations with this gas. Sulphuretted hydrogen appears to act like a narcotic poison when highly concentrated, but like a narcotico-irritant when much diluted with air.

Irritation of the mucous membrane of the eyes, nose, larynx and air tubes is found in subacute cases and the patient may have severe fits of coughing, acute bronchitis, broncho-pneumonia or œdema of the lung. Severe inflammation of the conjunctiva may occur.

In the chronic cases occurring amongst the Thames Tunnel workmen the symptoms were thus described by Taylor:

"As a result of breathing this atmosphere the strongest and most robust men were, in the course of a few months, reduced to an extreme state of exhaustion, and several died. The symptoms with which they were first affected were giddiness, sickness, and general debility; they became emaciated, and fell into a state of low fever, accompanied by delirium. In one case, the face of the man was pale, the lips of a violet hue, the eyes sunk, with dark areolæ round them, and the whole muscular system was flabby and emaciated. Chlorinated lime and other remedies were tried for the purification of the air; but the evil did not entirely cease until the tunnel was so far completed that there was a communication from one side to the other, and free ventilation established throughout."

Treatment. An immediate removal to pure air, artificial respiration and the application of stimulants, with cold affusion, may suffice to restore life in acute poisoning. In chronic cases means must obviously be taken to purify the air, and rescuers must work for only very short periods in the atmosphere or wear efficient respirators.

Post-mortem Appearances. The gas is *absorbed* into the blood, to which it gives a brownish-black colour by reducing the oxyhæmoglobin to hæmoglobin and finally forming SH_2-hæmoglobin, and it is in this state circulated throughout the body. In moderate concentrations, however, it fails to form sulphæmoglobin, and the hæmoglobin, of course, recombines with oxygen, so that no changes in the blood pigment are found. On examining the bodies of persons who have died from the effects of sulphuretted hydrogen, when breathed in a concentrated form, and the inspection was recent, the following appearances have been observed: The mucous membrane of the nose and throat is commonly covered by a brownish viscid fluid. An offensive odour is exhaled from all the cavities and soft parts of the body. These exhalations, if received into the lungs of those engaged in making the inspection, sometimes give rise to nausea and other unpleasant symptoms, and may even cause syncope or asphyxia. The muscles of the body are of a dark colour. The lungs, liver, and the soft organs generally, are distended with black liquid blood, and the blood has been found everywhere liquid and dark-coloured. The body rapidly undergoes putrefaction. When death has

occurred from the inhalation of this gas in a more diluted form, the appearances are less marked. There is then general congestion in the internal organs, with a dark and liquid state of the blood. In fact, in such cases the appearances can scarcely be distinguished from those produced by carbonic acid. Four men lost their lives in the Fleet Lane sewer in February 1861: they were found dead; and there was no doubt that sulphuretted hydrogen was the cause of death. An account of the appearances presented by the bodies was given by Holden and Letheby.[1] The eyes and mouth were open, the lips and tongue livid, the pupils widely dilated, the blood black and fluid, the lungs congested, the heart full of black fluid blood, the right side gorged, and there was a bloody froth in the windpipe. In the brain the large vessels of the dura mater were full of black fluid blood.

Except for the smell there are no diagnostic features in the *post-mortem* appearances.

Sulphæmoglobin may be looked for. If a sample of blood is diluted so that only the red part of the spectrum remains visible, an absorption band in the red is evidence that sulphæmoglobin or methæmoglobin is present. In the case of the former pigment, the band is unaffected by addition of a reducing agent like sodium hydrosulphite or ammonium sulphide, whereas methæmoglobin is reduced by these substances with disappearance of the band.

Analysis. The detection of the gas by the smell is the best test, but corroborative tests may be applied by obtaining the sulphides of three or four metals from solutions of the gas by addition of soluble salts of the respective metals—*e.g.*, lead, copper, silver, antimony. It is, of course, essential to remember that hydrogen sulphide is a normal product of putrefaction.

In air hydrogen sulphide may be detected and determined quantitatively by slowly drawing a measured volume of the air through test paper impregnated with lead acetate, the brown stain produced being then compared with standards.

Cases. In 1857, six persons lost their lives at Cleator Moor by the respiration of sulphuretted hydrogen in a diluted form, by reason of their having slept in small close non-ventilated rooms, into which the gas had penetrated. Three of the deceased persons—a husband, wife and child, of one family—had retired to rest in their usual health. Two of them were found the next morning dead in bed, and a third (the child) was found in a state of insensibility, and lingered until the afternoon of the same day, when she died. The fourth, a healthy adult, retired to sleep in his bed, with his door closed, and he was found dead in *an hour*. The fifth, a child, was taken ill on the morning of the 11th, and died the same day. The sixth was taken ill on the morning of the 10th and died on June 12th.

In one child the pupils were found dilated, viscid mucus escaped from the nostrils, and there was congestion of the lungs and kidneys, as well as of the membranes of the brain. In the adult who died in an hour, the pupils were natural, the jaws firmly clenched, the fingers contracted, and the nails blue; there was great cadaveric lividity, and a quantity of fluid with frothy mucus issued from the nostrils and mouth. The lungs were much congested, and serum was effused in the cavity of the chest. The heart contained a little fluid blood, and was somewhat flaccid. The mucous membrane of the windpipe and gullet was redder than natural. In the windpipe there was frothy mucus. The stomach, as well as the large and small intestines, were highly congested, but otherwise healthy. The brain and its membranes were greatly engorged with blood, which, as in the body generally, was

[1] *Lancet*, 1861, 1, p. 187.

very dark and fluid. Wilson, who examined the body of the child, drew the conclusion, which was confirmed by the subsequent inquiry, that death had been caused by sulphuretted hydrogen. Thompson, who examined the body of the man, also inferred that some noxious gas or gases had destroyed life. The cottages in which the accidents had occurred were built upon a heap of iron-slag which also abutted on the premises behind. This slag contained, among other matters, sulphides of iron and calcium. A foul smell, compared to that of cinders extinguished by water, had for some time been perceived about the rooms, chiefly at night, when the doors and windows were closed; and the day before the occurrence a heavy storm of rain had washed through the slag-heap, and aggravated the effects. The heap of slag was burning in certain parts, and sulphuretted hydrogen was evolved in large quantities at a depth of a few feet. A fortnight after the deaths, on removing the flags in the lower rooms, the slag below was found damp, and sulphuretted hydrogen was still issuing from it. The white-lead paint in the closets was partly converted into black sulphide, and this chemical change was found in patches on the chamber door of one small room in which two persons had died.

The symptoms, so far as they were observed in the survivors, the appearances in the dead bodies, and the chemical nature of the wet slag beneath the foundation, left no reasonable doubt that during the night, with the doors and windows closed, sulphuretted hydrogen had escaped in sufficient quantity to poison the air of a small room and destroy life; and a verdict was returned to this effect. A suggestion was made that carbonic acid might have caused the symptoms and death, but there was no source of carbonic acid but the breath; and there is no instance known of any adult having breathed himself to death in an hour, in a room containing 600 cubic feet of air—not to mention that persons had slept in similar rooms in the same row of cottages, at a distance from the slag-heap, without perishing from such a cause. Another theory was put forward to the effect that carbonic oxide in the vapours of some blast-furnaces had found its way into the rooms where these persons had died; but the nature of the locality and the distance of the furnaces rendered this impossible. Persons who had left their windows open, whereby these vapours might have freely entered, escaped, while the deaths occurred only in those houses in which the doors and windows were completely closed. It is highly probable that the sulphuretted hydrogen was mixed with other gases and vapours; but the circumstances left no doubt that it was the principal agent of death. This seems to have been clearly established by the fact, that after a channel had been cut through the slag-heap, and the slag removed, no further accidents occurred.

The following case is unusual in the temporary recovery followed by death:—

While the students at the Industrial Institute of Lille were engaged in the determination of chemical salts in the Laboratory on Wednesday last, M. Beaubois, aged twenty-two, opened the apparatus in which the sulphuretted hydrogen was being prepared, and immediately fell suffocated to the ground. He was, however, able to get up in a few seconds, went out into the fresh air, and soon felt better. M. Beaubois had hardly left the laboratory when M. Giraud, aged eighteen, also fell to the ground, although, as it appears, he was standing several yards away from the apparatus. His fall was so violent that it was thought that he had been killed, and he was carried out into the courtyard, where however, he soon regained consciousness. During more than an hour he walked about the court with the help of his friends, and appeared to have quite recovered. Towards three o'clock in the afternoon, however, he fainted again, and remained unconscious, apparently asleep. The doctor was sent for, and ordered his removal to the hospital, where his case received the greatest attention. He failed, however, to regain consciousness, and died at one o'clock on Friday morning.[1]

Poisoning by Sewer-gas and Exhalations from Dead Bodies

Source and Method of Occurrence. This is sufficiently explained by the title. The cases are always accidental, though it is easily conceivable that a suicide or a homicide might thus be effected.

[1] *Pharm. Jour.*, November 1899.

In 1863 two persons were killed by the effluvia which had collected in one of the deep graves kept open in Aldgate Churchyard. With ordinary precautions, and the use of deodorisers, the remains of the dead may be removed and transported to other localities without injury to the living. Within a few years many bodies were thus removed, without ill effects, from London cemeteries, as those of St. Andrew's, Holborn, and St. Pancras.

About the latter part of the 18th century, from fifteen to twenty thousand bodies, in almost every stage of putrefaction, were removed from the Cimetière des Innocents in Paris; and the accidents which occurred during the operations, which lasted ten months, were, comparatively speaking, few. The workmen acknowledged to Fourcroy that it was only in removing the recently interred corpses, and those which were not far advanced in decomposition, that they incurred any danger. In these cases the abdomen appeared to be much distended with gaseous matter; when ruptured there issued a bloody fœtid liquid, accompanied by the evolution of a mephitic vapour—probably a mixture of carbonic acid and sulphuretted hydrogen. Those who breathed this vapour, as it escaped from the body, fell instantly into a state of insensibility and died; whilst others who were at a distance, and who consequently breathed it in a diluted state, were affected with nausea, giddiness or fainting, lasting some hours, and followed by weakness and trembling of the limbs. Some years ago, when it was the practice to bury the dead in the crowded churchyards of London, lives were frequently lost by reason of the noxious gases and effluvia which at once filled every grave as it was made. A grave twenty feet deep was usually dug between strata of exposed coffins, and this grave was kept open until it was filled with bodies.

Dr. Haldane says, "besides sulphuretted hydrogen, almost the only gas likely to be present in poisonous proportions in sewer air is the carbon monoxide of coal-gas. It is well known that coal-gas from leaks in the mains very frequently penetrates into sewers, and occasionally causes explosions. It can usually be recognised by its smell, though this may be absent if the gas has passed through fresh earth, or may be disguised by the smell of sewage. If more than about 5 or 6 per cent. be present, the air of the sewer will be explosive; and as sewer-men must always be provided with lights, the poisonous action of more than 5 per cent. need hardly be considered. Now, as coal-gas itself contains only about 5 per cent. of carbon monoxide, air containing 5 per cent. of coal-gas will only contain about 0·25 per cent. of carbon monoxide. The experiments which I recently described in the *Journal of Physiology* show that 0.25 per cent. of carbon monoxide would be at any rate sufficient to render a man absolutely helpless, so that if in a sewer he would probably be drowned. It would, however, require at least half an hour's exposure to cause any danger. Even with as little as 0·06 per cent. of carbon monoxide, or 1·2 per cent. of coal-gas, a condition would be produced, after an hour or two, in which any extra exertions, such as that of climbing up a manhole, might give rise to temporary loss of consciousness or giddiness and thus easily cause a fatal accident. There is, therefore, no doubt that coal-gas may be a source of serious danger in sewers, quite apart from its explosive properties, and probably some cases of poisoning have been due to it, though so far as I know no such case has been recorded. An observation of the colour of the blood when diluted to $\frac{1}{4}$ with water, and compared in a test-tube with the colour of a similarly diluted solution of normal blood, would always decide the question" (Haldane, *l.c.*).

For a report on the general effects on health of sewer air, *vide Lancet*, 1909, I, p. 1402.

Toxicity and Fatal Dose. The fatal dose is quite unknown. As regards the toxicity of sewer gases, while SH_2 seems to be the most noxious, the rest cannot be considered to be free from suspicion, and it is on this account that sewer-gas poisoning receives some notice apart from SH_2 and CO and CO_2. Dr. Haldane, in the article below, says :

" Wherever sulphuretted hydrogen is formed in sewage, marsh gas is probably also formed in far greater amount." The possibility of suffocation from simple deficiency of oxygen in the air of a sewer must also be taken into account.

In the *Lancet* for January 25th, 1896, p. 220, will be found a long article on the subject by Dr. J. S. Haldane, Lect. in Physiology, Oxford, of which the following is a summary. " On July 21st, 1895, five men were killed in an accident at the East Ham Sewage Works. The air in the manhole smelt very strongly of decomposing sewage, but I had frequently been in sewers in which there was an equally unpleasant smell. I was unable to detect in myself any unusual symptoms. A careful analysis of the sample of air gave the following result : oxygen, 20·84 per cent.; carbonic acid, 0·10 per cent.; and nitrogen and argon, 79·06 per cent. As with the same gas-burette fresh air gave 20·91 per cent. of oxygen, it follows that the oxygen in the sample was diminished by 0·07 per cent., and the carbonic acid increased by the same amount. Thus, according to the analysis, the air differed very little from normal air, and was only slightly less pure than that of average well-ventilated sewers such as those investigated by the late Professor Carnelly and myself. The slight vitiation detected by analysis would have required to be more than a hundred times as great to produce any serious action on a man. The result of the examination of the air thus threw no light on the cause of the accident, and was, at any rate, unfavourable to the theory that there had been any dangerous deficiency of oxygen or excess of carbonic acid in the air. The live coals lowered into the manhole at the time of the occurrence burned perfectly brightly, and this fact pointed strongly in the same direction. Flames are extinguished when the proportion of oxygen in the air is diminished by 3 or 4 per cent., though this diminution causes no inconvenience to a man. It is true that if pure carbonic acid be added to air a flame will still burn in it until the atmosphere is such as to be capable of causing extreme respiratory distress and other serious symptoms in a man breathing it ; but such an admixture of pure carbonic acid would not occur in a sewer."

Dr. Haldane then proceeded to prove by experiments with the sewage itself and a mouse that the symptoms were due to SH_2. He then discusses the liberation of SH_2 from sewage and its accumulation to a point of danger in the air, which point he puts at 0·07 per cent. on the authority of Lehmann, who says[1] : "This percentage is just sufficient to cause death after an exposure of about an hour or more in the case of various animals, and doubtless also in man." Lehmann is further quoted : " In one of his experiments on man alarming symptoms were produced within a few minutes by an atmosphere containing about 0·05 per cent. The frequently quoted statement on Parent Duchatelet's authority that a man can breathe 3 per cent. of sulphuretted hydrogen for a short time without injury is certainly quite erroneous ; 3 per cent. would cause almost instantaneous death. Lehmann found that 0·2 per cent. was sufficient to kill dogs and cats within one and a half minutes. Lehmann's experiments on man brought out clearly and for the first time the symptoms which are produced in men and animals when the proportion of sulphuretted hydrogen is becoming dangerous. The first symptoms are those of irritation of the eyes and respiratory mucous membrane. In consequence of the irritative action on the air passages there is ' catching of the breath ' and diminished frequency of the respiration. With increasing

[1] " *Arch. für Hyg.*," 1892, p. 135.

proportions of the gas or longer exposure these symptoms are accompanied by giddiness, etc." Although in the case of the East Ham accident the poisonous constituent in the sewer-air was sulphuretted hydrogen, yet it does not follow that cases of acute gas poisoning in sewers are always due to the same cause. After examining the evidence in several cases, however, Taylor was inclined to think that they usually are. On May 6th, 1894, two men were knocked down by gas and drowned in a sewer under the control of the Lambeth Corporation. The case was very thoroughly investigated at the inquest. It appeared that three men who had been sent in to measure a sewer were suddenly overcome by gas and fell into the water. Two were drowned, but the third regained consciousness, and succeeded with great difficulty in escaping. The sewer was an old and filthy one, containing much deposit; and it appeared that a quantity of warm water containing about 1 per cent. of sulphuric acid was daily discharged into it from an oil refinery. The man who escaped did not notice the smell of rotten eggs, but observed steam coming down the sewer, and then suddenly experienced a choking sensation and lost consciousness. His silver watch and chain were blackened. A man who afterwards attempted to rescue the other men noticed a smell which resembled that of rotten eggs. He also experienced the choking sensation; he became giddy, and had to return. Another man, who went in later, said that the gas made his throat feel dry and his eyes smart. He described the smell as oily. A sample of sewage taken at the same place next day was analysed by an experienced analyst who found in it 0·13 per cent. of free sulphuric acid and 10 per cent. by volume of sulphuretted hydrogen. Dr. Stevenson expressed the opinion at the inquest, that the accident was due to dilute sulphuric acid coming in contact with sulphides present in the sewage and thus liberating sulphuretted hydrogen. An accident in the Fleet Lane sewer in 1861 was attributed by Letheby to a similar cause, although the presence of acid was not proved.

Duration. The above discussion shows quite conclusively that, whatever be the cause of the symptoms, they occur very quickly indeed after exposure to the fumes, and death follows within a few seconds or at most a few minutes, as a rule, though some of those who recovered would seem also to have been exposed for a much longer time. When recovery takes place, it is generally complete in about twenty-four hours, though in the *B.M.J.*, 1896, 1, p. 269, chorea is alleged to have followed a non-fatal case.

Symptoms. All cases seem to be alike in the rapid onset of powerlessness, which in turn may lead to drowning, etc. In a case of multiple poisoning reported in the *B.M.J.*, 1903, 2, p. 845, it is said of the five men :—

"During their treatment in hospital the clinical features observed were very few, and nothing was discovered which throws any distinct light on the nature of the gas by which the men had been affected. Some of them vomited while in the surgery, and one after removal to the ward, and all of them were in a state of semi-collapse, sufficient in one case to render the use of oxygen desirable. When reaction set in they were all

of them very incoherent and drowsy, and remained so until next day, when some complained of much headache."

In Dr. Haldane's case the following symptoms are reported :—

"The rescued man was in a comatose condition when brought up, and never recovered consciousness. He died in the West Ham Hospital almost eighteen hours after. Mr. Blake, resident medical officer to the hospital, kindly furnished me with information as to his symptoms. When he was brought in his respiration was slow and stertorous. There was no corneal reflex. Distinct cyanosis was absent. There were constantly recurring clonic spasms of the arms and legs ; these spasms were repeated about every half-minute, and rendered it very difficult to carry out artificial respiration, which was employed for the first two hours. The eyes were frequently directed towards one side. The temperature rose to 102° F. some time after his admission. No symptoms of bronchitis were noticed. There was a discharge from the eyes indicative of conjunctivitis. Urine was passed involuntarily, and none could be collected. Inhalation of oxygen was tried, but without effect. Three hours before death the spasms ceased, and symptoms of œdema of the lungs appeared and gradually increased."

Treatment. Precisely the same as for CO, CO_2, and SH_2 in the pure form.

Post-mortem Appearances. Except for rapid decomposition there is nothing distinctive found. Dr. Haldane thus records the autopsy of the case he dealth with :—

At a *post-mortem* examination, conducted by Mr. Blake, Mr. J. H. Horton, and myself three days later, no fresh naked-eye changes of any kind were discovered in the organs, with the exception of the lungs, which were more or less œdematous. The blood was normal in appearance, and there were no haemorrhages or other gross changes in the brain or spinal cord, which were both examined. Unfortunately, decomposition was already far advanced. Dr. Washbourn and Mr. Horton made a microscopic examination of parts of nearly all the organs. They found patches of broncho-pneumonia in the lungs, but no distinctly recognisable fresh changes elsewhere. The brain, spinal cord, liver, kidneys, and heart were all examined. The body of one of the other men was also examined by Dr. Smith. The appearances were such as to suggest that death was due to drowning. I examined a specimen of the blood and ascertained that no carbonic oxide was present. There were no signs of any abnormality in the haemoglobin. Four men had thus been drowned while stupefied by the gas, while the fifth had apparently been killed by the gas alone.

Analysis. The odour of gases and vapours from sewers and from the dead is sufficient to determine their presence, even when they are diluted with a large quantity of atmospheric air. *Sulphuretted hydrogen gas* is identified by its action on paper previously dipped in a solution of salt of lead : if present, even in a very small proportion (1-100,000th part), the moistened paper speedily acquires a brownish-black stain from the production of lead sulphide. In a mixed atmosphere of carbonic acid and sulphuretted hydrogen, the two gases may be separated by agitating the mixture with a solution of acetate of lead and treating the precipitate with acetic acid, which dissolves the carbonate and leaves sulphide of lead.

Dr. Haldane (*loc. cit.*) remarks on the smell :—

"As regards the failure of some of the men to recognise the specific smell of sulphuretted hydrogen, it must be remembered that when it is present in relatively large quantities the smell is not nearly so characteristic. The sense of smell for sulphuretted hydrogen seems indeed to become paralysed, just as occurs in the case of skatol and other very strongly smelling substances. I have myself met with gas (coming from a heated coal-heap) which at once blackened lead paper, but in which I was quite unable to smell the sulphuretted hydrogen. The smell was quite perceptible, however, when I opened in the fresh air a bottle containing a sample of the same gas." CO- and SH_2-hæmoglobin may be looked for (*vide* above).

Cases. The following case reported by Dr. Haldane serves as a type :—

One of the men had gone down the ladder as usual to clear the accumulation from the front of the screen. Before reaching the bottom he said he felt ill and began to return ; but when almost at the surface he was overpowered, and fell off the ladder into the sewage, which seems to have been about four feet deep at the time. (It was a rule that the men were not to go down unless the top of the sewer was free, so as to allow of the ventilation being in operation. The ventilating shafts are connected with the crown of the sewer.) A companion who was assisting at the surface immediately summoned help, and with rash but splendid courage three men descended, one after the other, to the rescue. They were, however, all overpowered and fell off the ladder to the bottom. The engineer in charge of the pumping-station was then called from his office, and seeing one of the men lying on the top of the screen, at once descended to his assistance, but fell off the ladder and was drowned. A bucketful of live coals was now brought from the engine-house and lowered into the manhole. After it had been withdrawn a man went down with a rope round his body, but had to be drawn up again. Another man, also with a rope round him, next went down, and was able to keep his footing. He succeeded in getting a rope round one of the men who, having fallen on the top of the screens, had not been drowned and was still breathing, though quite insensible. The poisonous gas seemed now to have lessened in amount, and the bodies of the other men were soon recovered from the water.

In the following case the actual lethal agent seems to have been doubtful :—

"A curious case of sewer-gas poisoning occurred lately in connection with some sewerage work in Union Street, Southwark, which entails the substitution of a new for an old sewer. Part of the work is so far complete that the roadway is filled in again, and in this finished section some men had been engaged during the night clearing out odd bricks and accumulated building rubbish. About 7 a.m. they knocked off work and proceeded along the sewer to the manhole. When only a few yards from this, they seemed to have walked into an accumulation of sewer-gas, and one after another they dropped down insensible to the number of five. A sixth man, who was only partially overcome, managed to reach the manhole and shouted for assistance, which was quickly accorded by a party of navvies working close at hand. After an unsuccessful effort had been made by one of their number, another navvy, named Leonard Dallimore, got down, taking a rope with him, and, one by one sent the men to the surface. During the process he wore no handkerchief nor other face protection, and the time during which he was exposed is uncertain. On completing his task and emerging from the sewer he could only say that he felt 'queer,' and that he could not have remained down much longer. On being brought to the surface, artificial respiration was applied to the unconscious men by some men of the Fire Brigade, to which an alarm had been given, and as soon as signs of life returned the five men were removed to Guy's Hospital. Under treatment there they quickly recovered, and had all been discharged after three days. No blood

from any of the cases appears to have been examined, but spectroscopic examination of the urine of the patients did not afford any evidence of destruction of red blood corpuscles. This negative observation, so far as it goes, seems to contra-indicate sulphuretted hydrogen as the active constituent of the sewer-gas in this case. This is somewhat curious, for it is so constantly present in sewer-gas, and lethal in such minute quantity, that in descriptions of sewer-gas poisoning many writers of text-books confine themselves to detailing the effects of sulphuretted hydrogen alone. It is understood also that Dr. Stevenson went to see these cases, and from their appearance and rapid recovery expressed the opinion that the cause of their collapse in the sewer could not have been sulphuretted hydrogen. The precise cause of their condition therefore remains somewhat of a mystery."[1]

Poisoning by Arseniuretted Hydrogen (Arsine, SH_3)

Source and Method of Occurrence. The gas is not used commercially as such, but large quantities are sometimes produced in chemical works, by action of acids on metals containing arsenic. Arsine is formed during the decomposition of impure ferrosilicon, it may be evolved during the charging of accumulators and, generally, in any process where metals or ores contaminated with arsenic are treated with acids. All recorded cases are accidental. Dixon Mann[2] records five cases, two of which were fatal, and at Accrington in August 1900, ten cases occurred.[3]

The latter are thus reported by Dr. J. S. Clayton :—

"The process in which the men were engaged was the manufacture of zinc chloride from crude zinc oxide and hydrochloric acid. The zinc oxide was a waste material obtained from galvanising iron, and was, in fact, a galvaniser's refuse. The HCl was made on the premises, and admittedly contained arsenic. Unfortunately the firm made no quantitative analysis of these crude materials, but their chemist informed me that a sort of examination was made of both, as is their usual custom, sufficient to show them that less than 0·1 per cent. of As was found in the HCl, and much less than that in the Zn, so little, in fact, as to make the quantity impossible to determine. They also stated that so far as they themselves knew no other case of AsH_3 poisoning had occurred during the twenty years that this $ZnCl_2$ process had been going on. That other cases have from time to time occurred is however quite certain, very decided information on this point being obtainable from the medical practitioners in the district, though they were isolated and of a less intense character.

"On this occasion it appears that the men, for some reason, were eager to get through a certain amount of work by noon, and two or three extra hands were taken on. The vat was kept well going, and more fumes than usual may have been evolved. The day was warm and unusually sultry. The fumes could not get away, and as the crude zinc oxide probably had mixed with it a small quantity of zinc carbonate, from which CO_2 would be evolved, the tendency of the gas would be downward, AsH_3 itself being heavier than air. In addition to this tendency, there would undoubtedly be the powerful indraught exerted by the furnace through the open door. Instead of this free access to the air being a security, on this occasion it constituted a danger. As a matter of fact, the men working nearest to the vat on the platform, and furthest from the well, suffered least, those down below in the well the most. Of those on the platform there were three. One was severely poisoned, one slightly, and the third escaped entirely.

"With respect to the remainder, four men were engaged shovelling zinc oxide from a cart on to the ground area, and were at no time within ten yards of the vat. All of these suffered more or less severely, and three others were occupied in loading barrows from this and conveying it up the gangway to the vicinity of the vat. These also were poisoned severely, one with a fatal termination on the seventh day."

[1] *B.M.J.*, 1903, 2, p. 845.
[2] *B.M.J.*, 1896, 1, p. 1332.
[3] *Lancet*, 1901, 1, p. 392.

Leschke ("Clinical Toxicology," 1934, p. 73) states that about 300 cases have been published, and that at the tin works of Wilhelmsburg there were twenty casualties, with eleven deaths in 1931.

Glaister's "Poisoning by Arseniuretted Hydrogen" (Edinburgh, 1908) contains considerable information on this form of poisoning.

Toxicity and Fatal Doses. The gas is extremely poisonous, an amount corresponding to 0·01 mgrm. of arsenic producing severe symptoms. As to the dose necessary to be absorbed into the blood to produce death no exact determination exists, but it must be something very small. Leschke (*loc. cit.*) puts it at 0·3 to 0·6 gramme. It kills by destroying the red corpuscles of the blood.

Duration of exposure is an important factor, and it has been said that exposure for one hour to a concentration of 0·17 mg. arsine per litre of air (*i.e.*, 1 part by volume in 20,000) is dangerous, exposure for 12 hours to one-fifth of that concentration is fatal. These figures refer to isolated exposures, with repeated exposures no concentration of arsine can be regarded as harmless. (Leaflet No. 9: Methods for Detection of Toxic Gases in Industry. H.M. Stationery Office, 1939.)

Symptoms. In mild cases there is headache and nausea sometimes associated with vomiting and diarrhœa. The characteristic feature is the excretion of blood pigment which colours the urine a port wine colour. There may be pain over the kidneys and occasionally suppression of the urine occurs. Jaundice occurs after a day or two. Dr. Clayton recorded the following:—

"The symptoms in the ten cases here referred to were characteristic throughout, with the exception of one man who appeared to have absorbed a very small quantity of the gas, and who was correspondingly slightly affected. It is only necessary to quote one case as typical of the series, that of the foreman, who was engaged in emptying bags into the vat, and who was the most seriously affected of those at the vat. He was a robust, powerful man, and had been engaged at this particular work for sixteen years. If there can be such a thing as immunity from AsH^3 he should have afforded an example. He had been at work there all the morning, and left his work at 2 p.m. At 2.30 he felt sick, nauseated, and depressed, with a hot, burning pain from his throat to his stomach, and with an intense thirst. This was soon followed by violent vomiting, at first of food, then of everything as soon as swallowed, even iced water. This again was followed by an equally severe diarrhœa: the discharges were at first loose faecal matter, then rice-watery, and finally contained blood. Added to this, there was haemoglobinuria and a rapidly developing jaundice, which within twenty-four hours assumed an intense coppery hue. As is usual in these cases, and without any knowledge of the surrounding circumstances and the obvious cause, a diagnosis of cholera would have been pardonable within the first twelve hours. The whole effect was that of an irritant poison taken by the mouth, being evidence that the arsenic in the gaseous state was absorbed by the blood direct from the lungs, and in its passage through the walls of the stomach was separated there and acted directly. Doubtless also some may have been swallowed in the saliva. The feeling of depression deepened into extreme prostration, the features were shrunk and cyanosed, the pulse thready, and the voice lost. These severe symptoms lasted with gradually diminishing severity for several days. The feeling of prostration, the anæmia, the almost pale green colour of the skin which supervened on the disappearance of the jaundice, lasted for several weeks longer, and it was only after the lapse of five weeks that he was able to return to work.

"In the one fatal case the symptoms showed no variation from the above, excepting that there was no tendency to recovery. Towards the close there was suppression of urine, and after thirty six hours of complete unconsciousness, he died on the seventh day.

" In all of these cases there was jaundice. In eight of them it was intense. In two only could it be described as slight. In nine there was also haematuria. Intense thirst and a burning pain in the chest characterised the onset and earlier stages of all the cases. Diarrhœa was troublesome in five, and all the cases, with the single exception referred to, suffered from profound anæmia in the later stages of the disease. The effect on the nervous system was more or less severe in nine. In six of these the prostration and collapse of the earlier stage was very severe.

" One man who was engaged with these ten, and who was on the platform nearly the whole time, escaped without any symptoms whatever, and the varying degrees in which they were affected would seem to indicate that individual susceptibility played an important part. The man who died could not have been described before this as a good life. He had been a heavy drinker, and the effects of chronic alcoholism no doubt militated against any tendency to recovery."

Treatment. Blood transfusion is essential owing to the destruction of red cells. Other treatment is symptomatic and must be directed to the damaged condition of the kidney and the effect of arsenic on the nervous system.

Analysis. If required must be that of arsenic, *q.v.*

Arsine may be detected and approximately estimated in air by drawing the air through test paper impregnated with mercuric chloride and comparing the yellow colour produced with standards.

REFERENCES

Glaister, " Poisoning by Arseniuretted Hydrogen from Scientific and Industrial Operations." *Trans. Int. Cong. Med.*, Lond., 1914, Sect. XIX.
Hegler, " Sammlung von Vergiftungsfällen," 1931, II, 205.

Caisson Disease or Poisoning by Compressed Air

Source and Method of Occurrence. The development of engineering skill has led to this trouble being added to a list of diseases to which workmen are exposed.

The disease can hardly, perhaps, be called poisoning, and yet there is no other place for introducing the subject into a work on medical jurisprudence.

The cases are obviously all accidental, and the only reason for discussing them is that claims for compensation for injuries thus sustained may be brought into court, and medical evidence may be required.

Attention was first drawn to Caisson disease when Triger (1841) used pneumatic caissons for sinking coal shafts through wet quicksands at Châlons. He forced iron tubes 4½ feet in diameter through the soil to a depth of 23 feet, introducing compressed air to drive out the water. Since that time Caisson work in compressed air has been carried out on an immense scale in connection with the construction of bridges, tunnels under water and the building of harbours, and until the last few years the work was associated with a great deal of sickness among the workers. Thus in 1859, in the construction of the Rhine bridge at Kehl, there were 133 cases of Caisson disease; in 1873, at the Brooklyn Bridge, 110 cases in four months; in the construction of the bridge over the Eider 380 cases; in 1885, in the Hudson Tunnel, 2 per cent. of the workers died per month; at Cernavoda 154 cases; and at the Nussdorf Works on the

Danube, in 1895, there were 320 cases which were studied by Heller, Mager and von Schrötter in their monograph, "Luftdruck Erkrankungen" (Wien, 1900).

In New York four great tunnel systems have been built under the East and North Rivers by means of compressed air. Between 1906 and 1908 four 23-feet tunnels were driven under the East River at a distance of 6,176 feet, this being so far the greatest undertaking yet carried out by compressed air, the pressure averaging + 32 pounds and occasionally + 42 pounds. About 1,000 men were daily at work in the headings, and there were 3,592 cases of sickness among the 10,000 men employed (= 36·92 per cent.). Twenty deaths occurred.

Pathology of the Condition. The cause of Caisson disease was proved by Paul Bert in his great work. "La Pression Barométrique," 1878, to be the setting free of bubbles of *nitrogen* in the blood and tissues during and after decompression. This has been so completely confirmed by Heller, Mager and von Schrötter, by Leonard Hill in association with his co-workers Macleod, Ham, Greenwood at the London Hospital, and by Haldane, Damant, Boycott and others, that any other theories possess only historic interest. Bert exposed animals to high pressure in an atmosphere poor in nitrogen and rich in oxygen and decompressed them quickly. In this case no nitrogen bubbles were found in the blood, for the oxygen is rapidly absorbed by the tissues. It is the *partial pressure* of nitrogen, not the atmospheric pressure, which is the cardinal factor in the production of compressed air illness. In striking contradistinction to oxygen pressure is the appearance of animals suddenly decompressed after exposure to + 10 atmospheres of air. Bubbles of gas are seen in all the vessels, the blood froths in the arteries and veins and even in the placenta and fœtus if the animal is pregnant. If the animal is slowly decompressed the bubbles are few and small and may be completely absorbed and produce no symptoms.

Dr. Wainwright[1] draws attention to the fact that there are two separate groups of symptoms: those which occur on entering and those on leaving the high pressure conditions. It is certain that the former depend on the rather sudden change in pressure on the middle ear. It consists of buzzing in the ear and a certain amount of a sensation of giddiness. These symptoms are quite transitory, and on their subsidence the men are able to continue at work.

Duration. The initial symptoms mentioned above come on at once and pass off in a few minutes. The more serious symptoms, mentioned below, are also fairly rapid in onset, *i.e.*, within a few minutes to half an hour or longer; their severity and their permanence depend largely on the locality of the local lesion. The effects of this lesion need not be apparent at once.

Symptoms. Von Schrötter and his fellow-workers classified the symptoms and cases in the following table and showed that the symptoms generally came on half an hour, at most six hours, *after decompression*. The higher the pressure the greater the number of cases.

[1] *Lancet*, 1900, 2, p. 1792.

	No. of Cases	Percentage
Ear trouble, pain, hæmorrhage	68	21·5
Myalgia	105	32·8
Arthralgia	60	18·8
Affection of the trunk, girdle, pain, chest pressure ..	10	3·1
Monoplegia	17	5·3
Paraplegia	26	8·1
Menière's Syndrome	14	4·4
Apoplectiform deafness	2	0·6
Vertigo	4	1·3
Aphasia	1	0·3
Asphyxia	13	4·0

Keays, who was in medical charge of the work in New York, has analysed the cases as follows :—

(*A*) Pain in various parts of the body, "bends," 3,278 or 88·78 per cent.

(*B*) Cases with pain also having local manifestations, 9 or 0·26 per cent.

(*C*) Cases showing pain and prostration, 47 or 1·26 per cent.

(*D*) Cases with symptoms referable to the nervous system :

1. Brain (hemiplegia), 4 or 0·11 per cent.

a. Sensory disturbances	36
b. Motor disturbances	34
c. Sensory and motor disturbances	10
	80 or 2·16 per cent.

(*E*) Vertigo, "staggers," 197 or 5·33 per cent.

(*F*) Dyspnœa and sense of constriction of chest, "chokes," 60 or 1·62 per cent.

(*G*) Partial or complete unconsciousness with collapse, 17 or 0·46 per cent.

Fatal cases.	Group *C*	6
	,, *D*	5
	,, *G*	9
		20 or 0·54 per cent.

Von Schrötter has summarised 137 fatal cases (1854–1897) and places them in two groups.

1. Those which exhibited central nervous symptoms and died from secondary complications after days, weeks or months. These included thirty-six cases, on twenty-six of whom autopsies were made.

2. Those who died soon after decompression. These were seventy in number with twenty-seven autopsies eighteen of which were of value for analysis.

Pain often very severe in one or more of the extremities or epigastrium ; this may or may not be associated with nausea and vomiting. Headache, vertigo, and unconsciousness are also common, and paralysis more or less extensive and complete. In rare cases sudden death, almost without symptoms, occurs.

It is easy to see how the paralysis will vary according to the severity and the situation of the local damage produced by blocking of small blood vessels. Indeed, the sudden deaths are equally easily explained by a severe brain lesion in one of the vital centres.

Treatment is essentially prophylactic and relates to the process of decompression and the method of its application. As Dr. Hill points out, "the increased proportion of nitrogen taken up by the blood in compressed air passes to the tissues which contain fat and water. About 66 per cent. of the body weight is water, and an average man contains 15–20 per cent. of fat. If we take 0·9 per cent. as the co-efficient of solubility at body temperature for the watery part and rather more than five times this amount for fat (Vernon), and suppose a man of 60 kilos has 15 per cent. of fat, it follows that there will be 970 c.c. of N dissolved in his body at 1 atmosphere, or roughly 1,000 c.c. At 2 atmospheres there will be 2,000 c.c.; at 3 atmospheres 3,000 c.c.; at 4 atmospheres 4,000 c.c., and so on, if he is exposed long enough for the tissues to become saturated. Experience shows that the liability to Caisson sickness increases with the duration of the compression, *i.e.*, that the absorption of excessive N is a gradual process."

At the St. Louis Bridge Works when work was carried out at 110 feet (+ 48 lb.), twelve men were killed and eighteen seriously injured out of a total of 352. When the length of shift was reduced from four hours to one no more fatalities ensued. Divers often come up from great depths in three or four minutes and may suffer no inconvenience provided they stay down a short time, *e.g.*, a minute or two. The famous diver Alexander Lambert salved 70,000*l.* worth of gold at a depth of 27 fathoms, descending thirty-three times. He usually stayed twenty to twenty-five minutes below, taking two or three minutes to go down and four or five to come up. In his last descent he stayed down forty-five minutes, and half an hour after coming up he became completely paraplegic.

With regard to decompression there are two views, both agreeing, however, that it must be slow. Dr. Leonard Hill and his co-workers consider that the decompression should be gradual and continuous, whereas Dr. Haldane recommended decompression in stages.

From the practical point of view, then, we have two methods of prophylaxis: (1) definite prevention of absorption by short shifts; (2) slow liberation by gradual decompression.

The treatment of symptoms when they occur under decompression is recompression, and this has been carried out in some engineering works, as at the Brooklyn Bridge and the Hudson Tunnel. At the East River Tunnel Works Keays found that recompression relieved 90 per cent. of the 3,067 cases of pain, failing to give any relief in only 0·5 per cent. of the cases.

When symptoms occur later they are beyond anything more than palliative treatment, but nature in some cases brings a *restitutio ad integrum* by her own methods.

Post-mortem Appearances. From the pathology of the condition as given above it is obvious *à priori* that these must consist of (1) gas in the blood-vessels with the results noted in Vol. I; (2) microscopic or macroscopic results of blocking or rupture of vessels. This has been verified by numerous autopsies thus:—

In the autopsies of von Schrötter's first group were found lesions of the spinal cord (disseminated and localised myelitis, haemorrhages in the

meninges and cord) and secondary complications like cystitis, pyonephritis, bedsores, etc. Of the eighteen well-carried-out autopsies in the second group, eleven showed the presence of free gas in the circulatory system. In the other seven cases there was congestion of the lungs and liver. In eight of the cases reported by Keays free gas was found in the vessels.

The locality of any focus of damage must be quite a matter of accident.

For those who desire to pursue the subject in more detail the following references will be useful :—

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Poisoning by Carbon Disulphide (CS_2)

Source and Method of Occurrence. The substance is a colourless or nearly colourless liquid used freely in indiarubber manufacture, in the manufacture of artificial silk by the " viscose " process, and also in chemical laboratories as a solvent for oils and fats, sulphur, phosphorus, etc. Cases arise mostly from the inhalation of the vapour by workpeople in factories, but the liquid carbon disulphide has been taken by the mouth with divergent results. In a case reported by Lewin, death ensued in two hours after a dose of 15 grams, while Sapelier has recorded an instance where, with a view to disinfecting his digestive tract, a man took 7 grams a day for a month, suffering only from diarrhœa and a loss of 20 kilogrammes of weight (cited by Vibert).

Toxicity and Fatal Dose. In the above fatal case the dose was 15 grams or approximately 230 drops. Lehmann has shown that a concentration of CS_2 to the extent of 1·5 mgrm. per litre of air will cause severe symptoms.

Symptoms. In acute cases there is irritation of the mucous membranes, headache and vomiting. There may be giddiness, tremor and muscular weakness and cramps, followed by stupor. Delpech has described with great elaboration the results caused by the inhalation of the vapours of the disulphide in caoutchouc factories.[1] The results rarely ensue, except in close, ill-ventilated establishments. He states that in these chronic cases there is intense oppressive headache, extending from the bridge of the nose to the temples, giddiness, and that on going into an uncontaminated atmosphere a feeling of intoxication is experienced. Sometimes there is a period of excitement ; but in all cases there is at a later stage dullness, apathy, and often partial paralysis of speech. Sight and hearing are affected. There is great loss of muscular power, and anæsthesia. Cramps and fibrillary contractions of various muscles are rarely absent. The sexual feeling, at first increased, is eventually completely lost. A number of the workpeople suffer from vomiting, colic, and alternate constipation and vomiting. Flatus, having the odour of the disulphide, may

[1] " Nouv. Recherch. sur l'Intoxic. spéc. qui détermine le Sulf. de Charbon," Paris, 1860,

be eructed and passed by the rectum ; and the urine also not infrequently smells of the poison. About 95 per cent. of inhaled CS_2 is excreted unchanged in the expired air.

Rendu[1] reports a case of poisoning by bisulphide of carbon. The patient was a girl, aged fifteen, who for about a year had been employed in vulcanising caoutchouc balls. She had for some time experienced frontal pain and heaviness of the head, but till shortly before admission she had no other troubles. About a month before entry the pains became more violent, and were soon accompanied by a painful contraction of the masseter muscles, then by stiffness of the neck and vertebral column ; finally the legs and arms were also attacked. It was found that the contractions, though generalised, were nowhere complete, but the contraction of the masseters prevented the jaws from being separated. It was probably this latter fact which had provoked a severe attack of ulcerative stomatitis, due to accumulation of tartar between the teeth. The tendon reflexes, too, were found to be slightly exaggerated. The later symptoms, judging from the order of their appearance, were the result of a further action of the poison on the central nervous system, first on the medulla and cervical cord and eventually on the whole spinal tract. It may be suggested that the affection was in a measure hysterical, but against this view there are the facts (1) that there was no history, either actual or hereditary, of hysteria ; (2) that the contractions were incomplete and generalised, the limbs being the least affected, there being also no disturbance of sensation ; (3) that the usual stigmata of hysteria were entirely absent. Beyond the contractions there was no nervous phenomenon. Other cases of poisoning by carbon bisulphide have been recorded, notably by Delpech, but none of them showed such a firm contraction of the jaws, and in them the contractions always passed off after a few days.[2]

In one or two cases from the Silvertown rubber works, the symptoms closely resembled hysterical or neurasthenic conditions, *i.e.*, complaints of subjective nervous phenomena with no signs of organic nerve disease.

The following is taken from the *Lancet*, 1796, 2, p. 51 :—

" Dr. Stadelmann recently drew attention to certain peculiar symptoms observed in workmen employed in indiarubber factories, and he showed three patients who, after having worked for some weeks in those factories, were seized with giddiness, headache, tremors, drowsiness, loss of energy, and gradual impairment of vision. One of them, a man aged twenty-eight years, complained of xanthopsy, and objects moving in the street appeared to him as if seen through a cloud : he also had painful contractions of the muscles and an increasing difficulty in walking or even in standing. Another patient suffered from stammering and fear of walking in the dark, and a further remarkable symptom was anæsthesia of certain portions of the skin. Some patients declared that their food had a sulphurous taste, and in grave cases insanity ensued."

Analysis. The odour and inflammability of this liquid are often sufficient to identify it even in the smallest quantity.

Carbon disulphide reacts with diethylamine and copper acetate to give the violet copper diethyldithiocarbonate. A mixture of 2 ml. of 2 per cent. diethylamine : in benzene and 2 ml. of 0·1 per cent. copper acetate in absolute alcohol gives a distinct colour with ·05 mg. of carbon

[1] " Sem. Méd.," November 11th, 1891.
[2] *B.M.J.*, 1891, 2, Epit., p. 162.

disulphide. The test can be used to detect and estimate carbon disulphide in air by slowly bubbling a measured volume of the air (freed from H_2S by means of lead acetate, since H_2S gives a similar colour) through the reagent and composing the resulting colour with that produced by known amounts of carbon disulphide.

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Riegler, " Die nervöse Störungen bei CS_2 Vergift." *Ztscht. f. Nervenheilk.*, 1907, 33.

Poisoning by Ferro-silicon

Source and Method of Occurrence. When iron ore, quartz, coke and lime are mixed and heated together in an electric furnace ferro-silicon is produced. It is extensively used in the production of steel. Some varieties with as much as 50 per cent. silicon disintegrate into powder on exposure to air with the production of poisonous gases, including phosphoretted and arseniuretted hydrogen. In the preparation of ferro-silicon phosphide and arsenide of calcium are produced, and it is these substances which, in presence of water, evolve the poisonous gases above mentioned.

Phosphoretted hydrogen is stated to be so poisonous that air containing 0·02 per cent. of the gas is fatal to small animals within half an hour.

In January, 1905, fifty steerage passengers were made ill and eleven died of poisoning. There have been other similar occurrences, one of which was the subject of investigation by Dr. Copeman, Mr. S. R. Bennett and Dr. Wilson Hake, who suggested certain regulations for the safe transport of ferro-silicon.

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Group 4. VARIOUS NON-METALLIC ELEMENTS AND COMPOUNDS

Phosphorus is the most poisonous representative of this group, and has much more similarity to arsenic and antimony in its ultimate action than it has to any of the members of this " group 4".

Poisoning by Phosphorus (P)

Source and Method of Occurrence. Poisoning with phosphorus is not very frequent in England ; but on the continent of Europe this poison is often selected for the purposes of suicide or murder. In England and Wales the annual average number of deaths from phosphorus poisoning over the period 1931-1940, was six (five suicides and one accidental). In France, within a period of six years, there were 103 cases of poisoning with phosphorus which gave rise to medico-legal inquiry. This poison is most commonly obtained from rat-pastes.

At an international convention at Berne in 1906 it was agreed to prohibit the use of yellow phosphorus in the match industry, and since that time it has ceased to be used in the civilised world.

Some matches contain phosphorus trisulphide, which is only slightly poisonous; others contain red phosphorus; safety matches contain no phosphorus, but the igniting mixture on the box contains red phosphorus.

Phosphorus is not often used for homicidal purposes owing to its smell and taste, as well as its luminosity, which readily reveal its presence.

At the Norwich Autumn Ass., 1871,[1] a girl of eighteen was convicted of an attempt to poison a family. She put a vermin poison containing phosphorus into a teapot; when hot water was poured into it, the smell at once led to suspicion. Phosphorus, which had been taken from a pot of vermin-killer carelessly left about, was found in the teapot. The girl was convicted.

Caspar describes a case in which the luminous appearance of the poisonous food led to a suspicion of poisoning with phosphorus, and this was subsequently proved.[2]

A woman put a preparation of phosphorus into some soup, and gave it to her husband. He ate it in a dark room in the presence of some friends, and they noticed that the liquid as he stirred it was luminous.[3] In this way a person may be warned and a life saved.[4]

For suicidal purposes phosphorus is usually taken in the form of rat-paste, a compound of yellow phosphorus, fat, sugar, and flour, coloured with Prussian blue. Sir Thomas Stevenson found a sixpenny pot of this paste to weigh 157 grains, and to contain eight grains of phosphorus. The dose of phosphorus is about one one-hundredth to one-twentieth grain, but it is no longer in the Pharmacopœia.

Red or Amorphous Phosphorus. The substance, known under the name of allotropic phosphorus, is not possessed of poisonous properties. This fact, long since announced by Liebig,[5] has been confirmed by all subsequent experience. It has been given to animals in doses of thirty grains without causing symptoms of poisoning.

Toxicity and Fatal Dose. That phosphorus is a powerful poison is proved by two cases quoted by Christison. In one, death was caused by a grain and a half within twelve days; in the other, by two grains within about eight days. One to two grains is a fatal dose. It is absorbed from the small intestine and circulates in the blood as phosphorus. Absorption is aided by the presence in the stomach or bowel of any fatty matters in which phosphorus is soluble. The urine voided during life has been observed to be luminous, and organic compounds containing phosphorus (phosphines) are also found occasionally. The production of phosphorous acid, by its oxidation, may irritate the stomach and bowel; but the element itself acts as a direct irritant to mucous membranes, and leads to cloudy swelling and fatty degeneration of all cellular structures.

Chevalier refers to a case in which a dose of 2·3 grains proved fatal, and two other cases in each of which a dose of 4·6 grains destroyed life. The same writer quotes, on the authority of Lobel, the case of a mental patient who died from a dose of one-eighth of a grain.[6] This is the smallest fatal dose for an adult on record.

[1] *R. v. Fisher.*
[2] Casper, E. Tr., pp. 58–60 and 97–101.
[3] *Vierteljahrsschr. f. Gerichtl. Med.*, July 1864.
[4] See *Ann. d'Hyg.*, 1870, 2, 203.
[5] "Letters on Chemistry," p. 165.
[6] "Ann. d'Hyg.," 1857, 1, 422.

A woman, *æt.* 52, took in divided doses, in four days, about six centigrammes, or less than *one grain*, of phosphorus dissolved. The largest dose taken at once, *i.e.*, on the fourth day, is stated to have been three centigrammes, or nearly half a grain. Symptoms of pain and irritation appeared, and the patient died in three days. The gullet, stomach, and small intestines were found much inflamed.[1]

When phosphorus is dissolved in any liquid, or when it is finely divided, as in phosphorus paste, its action is then more powerful, as it is in a state well fitted for absorption. Kessler has recorded a case in which an infant seven weeks old was destroyed within four hours through swallowing the heads of six or seven matches, containing about one-eighth of a grain of phosphorus.[2] Sonnenschein refers to the case of an infant of five weeks who died from the effects of a single match-head, containing possibly not more than one-hundredth of a grain of the poison.

In 1882 a man, *æt.* 32, was admitted into Guy's Hospital, having taken half a sixpenny pot of phosphorus paste in whisky at 3 p.m. He had vomited before his admission at 5.30 p.m. An emetic was administered on admission, which acted immediately. The vomit emitted white fumes, and smelt strongly of phosphorus. He was pale, cold, depressed, and showed signs of alcoholism. His breath had a slight alliaceous odour. There was severe epigastric pain, with a burning sensation in the throat and gullet. The emetic was repeated; and he vomited at intervals till 2 p.m. The last vomit had no phosphoric odour. Large doses of carbonate of magnesium were given in mucilage and milk. He felt relieved of the pain in the epigastrium, though there was some tenderness on pressure. Twenty-four hours after the administration of the poison he began to take oil of turpentine, beginning with 15-minim doses; and this was increased subsequently to 30 minims. On the third day the liver began to enlarge, and on the fourth day there was decided jaundice. The liver continued to increase in size till the sixth day, when it began to decrease. He suffered from headache and drowsiness. When the liver began to decrease in size, the oil of turpentine was discontinued. The patient made a good recovery. At one time the liver dullness extended to the upper border of the fourth rib on both sides, extending to the left beyond a vertical line from the left nipple, and downwards it reached nearly to the navel.

The dose taken was probably four grains.

Duration. Symptoms are commonly delayed for from a quarter of an hour to three or four hours, and the total duration varies greatly, from a few hours to two weeks or more. In a case related by Orfila death took place in four hours. In another case, also related by him, death occurred only after seventeen days. Habershon quotes a case which is said to have proved fatal within half an hour.[3] This is the shortest period recorded. In general, several days elapse before a fatal result occurs, and during this time the patient undergoes much suffering. In two cases of acute poisoning with phosphorus communicated by Moore, one proved fatal within seventy-two, and the other within eighty-eight hours. In a case which occurred to Anderson, a child, aged one year and eight months, had sucked the heads off about twenty phosphorus matches before detection. No symptoms appeared until the second day, when the child was drowsy, and slept for twenty hours. On the fourth day it vomited, the skin was hot, the tongue was dry, there was great thirst, with a quick pulse and cold extremities. On the sixth day there was much vomiting of a matter like coffee-grounds (altered blood). There was great pain in the stomach—the child became unconscious and gradually sank, dying on the seventh day

[1] "Toxicologie," vol. 1, p. 87.
[2] *Vierteljahrsschr. f. Gerichtl. Med.*, N.F. IV., p. 271.
[3] "Med.-Chir. Trans.," 1867, vol. 50.

after taking the poison. There was no purging, but the motions were passed involuntarily, and contained coagulated blood. An alliaceous odour was perceived in the breath during the progress of the case, and the body had a yellowish (icteric) tint.

Modern research has with tolerable completeness explained these great discrepancies in the duration of a case of poisoning by phosphorus. In a case that ends fatally within a few hours the result is due in all probability to shock and exhaustion, possibly also assisted by slight absorption of the poison with an acute effect upon the heart. In the cases that end after several days, the fatal event is due to the power which phosphorus is now known to possess of inducing degenerative changes in gland and muscle cells, changes primarily and chiefly of a fatty character, but these changes take some little time to reach a fatal extent. They are found typically and visible to the naked eye in the liver and heart, but can also be demonstrated in the stomach, kidneys, and muscular tissues generally; they fully explain the hæmorrhages in organs and tissues, for they are found in the muscular coats of the arterioles.

Symptoms. In the first instance the patient experiences a disagreeable taste, resembling that of garlic, peculiar to this poison. An alliaceous or garlic-like odour may also be perceived in the breath. There is pain and oppression in the region of the stomach, malaise, eructation of phosphoric vapours, having a garlicky odour; and these may be luminous in the dark. Vomiting is sometimes frequent and violent; in other cases quiet and at longer intervals. The abdomen is distended. Purging is not common. The vomited matters are coffee-coloured, or yellow and bilious, and may be luminous. There is intense thirst. The symptoms may increase in severity, ending in death from collapse in the course of a few hours—four to eight in the worst case.

Nevertheless, in the majority of cases the progress to a fatal termination is slower and more insidious. The irritant symptoms in a great measure subside and though the pulse is feeble, and there is a certain amount of malaise, the patient may to a casual observer appear to be in almost a normal state of health. But after the lapse of three or four days, jaundice sets in and rapidly increases, there is great prostration of strength, the abdomen becomes distended, the liver is observed to be greatly enlarged, and vomiting of altered blood may come on with intense thirst; the skin is cold, and probably scattered hæmorrhages appear beneath it; the pulse becomes feeble, rapid, and perhaps imperceptible at the wrist; the urine is scanty and high coloured and contains casts from the kidneys, excess of ammonia, lactic acid, bile and some leucine and tyrosine. The fæces, previously suppressed, are now more abundant and contain blood. Coma sets in, with jactitation of the limbs, or muscular twitchings; and the patient succumbs generally about five or six days after the administration of the poison.

F. E. Elkins has described very fully the symptoms exhibited in a case of phosphorus poisoning by matches; and Middlemas made an elaborate microscopical examination of the tissues in the same case. Briefly stated, the symptoms and the physical signs were, in the order of their appearance:

Alimentary System. Nausea; retching; vomiting; "indigestion feeling"; disagreeable taste of "rotten eggs" and "rotten greens"; discomfort, then pain, and later spasmodic pain, in the hepatic region; increased hepatic dulness;

jaundice; intense thirst. *Circulatory System.* Increase of pulse rate; compressibility, and later irregularity and threadiness of pulse; palpitations; cardiac sounds indistinct and distant; cardiac failure and temporary stoppage of radial pulse; faintness; lividity; coldness of extremities. *Respiratory System.* Yawning; irregular respiration approaching the Cheyne-Stokes type. *Urinary System.* Urine scanty, high-coloured, of high specific gravity, with traces of albumen and bile, and having a deposit of urates, mucus, fatty casts, and *débris*, and leucine and tyrosine may be found in it. *Nervous System.* Mental symptoms: listlessness; drowsiness; restlessness; mental confusion; inability to understand what was said; inability to answer questions readily or correctly; inability to recognise friends; semi-consciousness; semi-delirium; delirium; fits of great restlessness and violence; constant use of the word "yellow" when delirious; maniacal expression and behaviour; unconsciousness; coma. Sensory symptoms: "rheumatic pains"; blindness (?). Motor symptoms: thick and drunken-like speech; pupils fixed and dilated; external strabismus on left eye.

The pathological interest of this case lies in the changes in the nerve cells of the cortex of the brain. The production of fatty degeneration shows that the tissues of the nervous system are not exempt from the powerful action in this direction which phosphorus possesses. The occurrence of the change in so many organs and tissues of the body would point to some fundamental alteration in the processes of metabolism which phosphorus has the power of bringing about.[1]

It may thus be said that there are four stages in phosphorus poisoning.

1. A latent interval between the swallowing of the poison and the onset of any symptoms varying between a quarter of an hour and two or three hours.

2. A period of irritant symptoms, vomiting and pain lasting several hours to a day or two, and ending either in death; or

3. A second latent period in which health seems to be restored and which may last for from about five or six days, the common period, up to as long as six weeks, in a case recorded by West.[2]

4. A period of symptoms closely resembling if not identical with those of acute yellow atrophy of the liver.

It will be perceived that, in reference to (1) the delay in the appearance of symptoms, (2) their similarity (taken as a whole) to disease, and (3) the time at which death occurs, cases of phosphorus poisoning might easily throw a practitioner off his guard when debating his diagnosis.

Treatment. The principles of treatment are, firstly, to evacuate from the stomach the swallowed phosphorus, and, secondly, to render harmless, if possible, that which is inevitably left behind.

For the first purpose the stomach tube should be used and the viscus thoroughly washed out. A weak solution of permanganate of potash (0·2 per cent.) should be used for this purpose, for it tends to convert phosphorus into phosphoric acid. This should be followed by the administration of large doses of animal charcoal. Intravenous saline is useful to combat shock, and the addition of alkali is indicated as the alkalinity of the blood is diminished. It is suggested that an emetic of copper sulphate (two or three grains) should be given, or that the stomach be washed out with a weak solution of copper sulphate, 3 grains to 8 ounces of water. This substance not only acts as an emetic, but also converts some of the phosphorus into copper phosphide, but care must be exercised lest the copper sulphate contributes to the gastric condition.

[1] *B.M.J.*, 1891, 2, p. 1302.
[2] *Lancet*, 1893, 1, p. 245.

For the second purpose all authorities are agreed that no oil nor fat should be given, for these dissolve the phosphorus, and therefore promote its absorption. Oxidised oil of turpentine and sanitas fluid have both been recommended, but apparently on very doubtful grounds.

Further treatment must be purely symptomatic (*vide* pp. 250 *et seq.*).

Post-mortem Appearances. These vary to a material extent according to whether death takes place within, say, twenty-four hours, or not till after the lapse of four or five days or more. In the former case the stomach and intestines show marks of irritation, inflammation, and ulceration. The stomach has been found much contracted, and its mucous membrane inflamed, occasionally softened and presenting purple or violet-coloured spots. Inflammation of the stomach and bowel may be a result of the action of phosphorus. A man, *æt.* 50, took a quantity of phosphorus paste used for destroying vermin. He was seen in his usual health at twelve o'clock at noon, and was found dead in a field the following morning. On inspection, it was observed that there was a great muscular rigidity. The heart was flaccid and nearly empty. The mucous membrane of the stomach, gullet, and small intestines was very red, and there were patches in which the membrane was destroyed. On opening the stomach a white vapour escaped, accompanied by a strong smell of phosphorus. This organ contained a tablespoonful of a viscid greenish matter, from which particles of phosphorus, with some Prussian blue (used as a colouring for the poison), subsided on standing.[1]

Schuchardt describes among the appearances, fluidity of the blood, which is of a dark colour, and does not become red on exposure to the air.[2]

In the cases with a longer duration the most remarkable appearance commonly met with is a fatty change in the liver, kidneys and other soft organs. Ecchymoses are often found beneath the skin and on the surface of various organs.

In the case of one female, who died after the lapse of a week, there was no inflammation, ulceration, nor softening of the mouth, gullet, stomach, or small intestines. There was a red patch in the cæcum, and another in the colon. The contents of the stomach and intestines had a coffee-ground colour, like the liquid found in hæmatemesis (vomiting of blood). There were bloody effusions in the chest and abdomen. The viscera, and even the flesh of animals recently poisoned by phosphorus, have the odour of garlic, and appear luminous in the dark. In one case, that of a girl, *æt.* 13, who died on the sixth day after taking phosphorus paste beaten up with egg, there were numerous ecchymosed patches in the cellular tissues of the skin of the abdomen over the rectus muscle: these were also seen on the chest and on the diaphragm. The stomach contained a dark-coloured thick fluid like altered blood; the coats were not inflamed: the surface of the inner coat was covered with a brownish-coloured mucus which had no odour of phosphorus. At the greater curvature the surface was dotted over with the numerous small dark particles, consisting of coagula of altered blood adhering to the membrane, but easily removed from it. The contents of the stomach owed their colour to these little masses of blood being diffused through them. The duodenum contained a similar liquid. The intestines presented no abnormal appearance.

[1] *Ibid.*, 1857, 1, p. 600.

[2] Von Hoffmann, Atlas, plates 47 and 48.

The liver was in an advanced state of fatty degeneration. In a case recorded by Habershon, in which a woman died on the fifth day, the symptoms and appearances were similar to those above described. The phosphorus was taken in the form of paste and, as was supposed, in a dose of from three to four grains. There was much ecchymosis in patches in and about the cellular tissue of the abdomen and chest. There was fatty degeneration of the liver and kidneys. The stomach contained a large quantity of fluid, like soot and water, and was covered with a tenacious bloody mucus. There was some congestion of the mucous membrane, and there was much redness with ecchymosis in the small intestines.

Sir Thomas Stevenson has seen decided enlargement of the liver produced within forty-eight hours of the time at which the poison was administered. The liver is usually enlarged, doughy, anæmic, and of a uniform yellow or yellowish-white colour. The acini are distinct. The hepatic cells are loaded with fat. The heart and muscles generally may be soft, yellow, and of defective tenacity. In place of the transverse striæ, innumerable fat globules are seen by the microscope. The glandular epithelial cells of the gastric follicles are filled with fat globules. The cortex of the kidneys is likewise filled with fat globules. For further information on this subject, see " Die Vergiftungen " by D. R. v. Jaksch, Leipzig, 1910.

In a case that died so short a time as fifty-nine hours after swallowing the poison the heart was found to have undergone fatty degeneration, and it and the aorta exhibited ecchymosed patches. The stomach was considerably injected, and its surface was thickly coated with tenacious mucus. The small intestine was much injected at its commencement, and to a less degree lower down. The liver weighed twenty-six ounces, was yellow, anæmic, and showed extreme fatty degeneration, except in isolated patches. In this case the dose of phosphorus could not be ascertained.[1]

Analysis. Yellow phosphorus, the poisonous form, is a waxy solid with a peculiar odour and taste reminiscent of garlic or celery. When exposed to air and light it evolves a white vapour, owing to its slow oxidation. In the dark it shines with a faint bluish light (phosphorescence). It melts at 45° C., and at about that temperature in air, it ignites, burning with a yellow flame and producing dense white fumes of the oxides. It is insoluble in water, but may be oxidised by oxygen dissolved in water. Hence water in which it has been kept contains various acids of phosphorus – hypophosphorous, phosphorous and phosphoric. It is readily soluble in carbon disulphide, and less easily so in alcohol, ether, chloroform, and various oils.

Red phosphorus is a dark red amorphous substance, which oxidises and ignites much less readily than the yellow allotrope. It does not exhibit the phenomenon of phosphorescence, and it is insoluble in carbon disulphide. The commercial preparation may contain small amounts (up to 0·6 per cent.) of the yellow form.

The following tests are designed to detect the poisonous yellow form of phosphorus.

[1] Guy's Hosp. Rep., 1877, 22, p. 449.

Scherer's Test (silver nitrate) is a useful preliminary test, but is most reliable when it gives a negative result—*i.e.*, a negative result indicates the absence of phosphorus, but a positive result merely indicates the presence of phosphorus or one of a variety of interfering substances. The test depends upon the fact that phosphorus vapour blackens a piece of filter paper wetted with silver nitrate solution. The most probable of the other volatile substances capable of doing this is hydrogen sulphide, and the test may therefore be carried out as follows. The suspected material is mixed with lead acetate solution (to fix the hydrogen sulphide) and made slightly acid with sulphuric acid. The mouth of the containing vessel is then closed by a cap of filter paper soaked in silver nitrate, and the vessel is gently heated. Amounts of phosphorus too small to be detected in this way may be found by more delicate tests, and in any case, a positive result requires confirmation.

Mitscherlich's test depends upon the fact that phosphorus vapour, as it condenses to the liquid form, is strongly luminous. The suspected material acidified with sulphuric acid is placed in a distilling flask connected to a water-cooled vertical condenser, the other end of which dips under water in a receiving flask. The whole apparatus must be in a perfectly dark room, and any gas or spirit burner used to heat the distilling flask must be carefully shielded (an efficient electric hot-plate is the best method of heating). The distillation is started and, in the presence of phosphorus, a ring of light appears, rising and falling in the condenser tube, at the point where the vapours are condensed. For quantitative work, about a third of the fluid may be distilled over, the collected phosphorus (part of which may be oxidised) converted to phosphoric acid by means of nitric acid, and then estimated by precipitation as magnesium ammonium phosphate or phosphomolybdic acid. Care must be taken, in this case, that no material from the distillation flask has been carried over mechanically.

The test can be carried out in the presence of most organic matter. Alcohol, ether, chloroform, and phenol prevent the appearance of the luminosity until they themselves have been distilled off; turpentine completely inhibits it, as do certain inorganic substances, such as calomel, corrosive sublimate, chlorine, sulphur dioxide and iodine. Ammonia also inhibits it (hence one reason for acidifying the fluid), as does hydrogen sulphide (which if present, may be fixed by addition of lead acetate).

Mukerji's test, also depending upon the production of phosphorescence (luminosity), has the advantages that no source of heat is required, and that it is not prevented by many of the substances which interfere with Mitscherlich's test (including hydrogen sulphide). It is, however, inhibited by turpentine or ether.

The apparatus consists of a three-necked Woulfe's bottle of about a litre capacity. One side neck is fitted with a long safety tube (passing through a tight-fitting stopper) reaching nearly to the bottom of the bottle; the other carries a short glass delivery tube drawn out to a jet; and the middle neck has a loosely fitting stopper through which passes a glass tube about 1 foot long and $\frac{1}{2}$ inch diameter, reaching just to the inside of the bottle, and closed at the upper end by a cork. Hydrogen is evolved in the bottle from zinc and dilute sulphuric acid. When the reaction has warmed the apparatus (which is placed in a dark room) and it has been

noted that the gas issuing from the jet is not phosphorescent, the suspected material is added. In the presence of phosphorus a sheaf of phosphorescence appears at the jet. If the middle tube be now uncorked, the light sinks down the delivery tube and appears at the top of the wide middle tube.

Separation from Organic Matter. When phosphorus is present in large amounts—*e.g.*, in samples of rat poison, or occasionally in vomitus or stomach contents—it may be separated by extraction with carbon disulphide. A few drops of the solution may be poured on filter paper and allowed to evaporate spontaneously—when evaporation is completed the paper ignites.

Duration in the Tissues. Phosphorus is slowly oxidised, and it must be remembered that, since phosphates are normal constituents of all tissues and most secretions, only the detection of elementary phosphorus is of toxicological value.

In one case of a girl who died on the sixth day, no phosphorus was detected in stomach or liver, nor did Sir Thomas Stevenson detect any in the stomach or stomach contents of Habershon's case, in which death took place on the fifth day. Herapath failed to detect phosphorus in a body on the twenty-third day after death. In poisoned animals, however, it has been found as late as ninety-seven days after death. Much must depend on the time which elapses between ingestion of the poison and death. It may occasionally happen that although phosphorus cannot be detected, colouring matter present in the poison may remain and afford a useful clue.

Cases. A female, *æt.* 20, took several doses of phosphorus paste; the first on the evening of January 11th, 1877. The dose was repeated twice on the 12th. The whole quantity of paste taken was of the size of a large cob-nut, containing about two grains of phosphorus. On the morning of the 13th she retched, and at midday her appetite failed at dinner; and in the evening she vomited. At 10 p.m. on the 14th she was first seen by Tyson, about seventy-two hours after the first and forty-eight hours after the last dose was taken. She had then an excited aspect, and her breath had a phosphoric odour. There was tenderness over the region of the stomach. On the 15th there was slight yellowness of the conjuctivæ of the eyes, slight pain over the stomach, and nausea, but no vomiting. The urine was high-coloured and turbid. On the 16th there was decided jaundice, great thirst, and prostration. There was still a slight garlicky odour of the breath; but the urine and faeces showed no luminosity. There was no obvious enlargement of either the liver or spleen. On the 17th the liver was enlarged; only a very little dark-coloured urine was passed; and there was much epigastric pain and tenderness. In the evening there was slight delirium. From this time she gradually sank, and died on the 18th, nearly a week after the administration of the first dose of the poison. On *post-mortem* examination the liver was found to be of the usual size; but it had undergone extensive fatty degeneration, as had the heart also. There were no marked appearances in the stomach, which was almost filled with a blackish syrupy liquid.[1]

In 1876 a woman and her daughter *æt.* 5, each drank some phosphorus paste in warm water. The woman was seen four days later apparently in her usual health. Subsequently she sickened, became jaundiced, and died a week after the poison was swallowed. The child exhibited no symptoms till 7 a.m. on the day after taking the poison. She then vomited some slimy material, and her breath had a garlicky odour. In a few hours she was in a state of semi-collapse. Next day she became drowsy, then thirsty, restless, and vomited constantly. There was no jaundice. She died fifty-nine hours after the administration of the poison.

[1] Guy's Hosp. Rep., 1877, p. 452.

In 1899 Caroline Davis, *æt.* 63, an inebriate, left drunk 8.30 a.m., and found at 8.30 p.m. (having probably taken red heads of matches) groaning, with abdominal pain, thirsty, and lying on floor. No vomit seen. Admitted into Guy's at 11.45 p.m. She was then collapsed, cold, especially in limbs. No pulse could be felt. Noisy breathing. Pupils medium. Slightly cyanosed. Sweating. Remained unconscious, but reacted to stimulants. Breath smelt of phosphorus. Reflexes present. Treated by stomach-pump, $KMnO_4$, stimulants, artificial respiration, and turpentine. Died at 1.15 a.m., four and three-quarter hours after she was found. *Post-mortem:* Slight congestion of gastric mucous membrane, and of upper part of small intestine. No match heads found. Liver fifty-two ounces, pale. Lungs very congested and œdematous (an old bronchitic). Epiglottis and trachea injected. No excoriation of tongue. Other viscera normal, except old thickening of meninges.

The following case, reported by Dr. Newry, *Lancet*, 1900, 2, p. 875, has features of considerable interest :—

On August 11th I was called to see a man who was suffering from vomiting and intense burning pain in the stomach and bowels. His history was that on the 7th, when "in drink," he had swallowed three pennyworth of rat-killer. Immediately after swallowing it he complained of violent pain in the stomach and sickness. He was given salt and hot water, after which he was very sick and vomited freely and expressed himself relieved. On the next day he was a little better, although he felt far from well; he, however, dressed himself and walked about. On the 11th he was taken much worse and I was sent for. When I saw him his temperature was normal, he was quite conscious and coherent, and his pulse was 90. He complained of thirst, constant vomiting, and great pain in the stomach and abdomen. An examination of the vomit proved it to consist wholly of altered blood of a very dark colour. His stools also were dark and pitchy in character. He was given the usual remedies, but they were of no avail, and he quietly sank and died on the following Tuesday, the 14th, having lived exactly a week after swallowing the poison. The vomiting of dark-coloured blood continued up to the time of his death.

A *post-mortem* examination was made twenty-four hours after death. The weather was very warm, but there were no signs of putrefaction. Very slight *rigor mortis*, if any, was perceptible. The body presented a most curious and interesting appearance. The whole of the neck in its entire circumference, back, and sides bore the appearance of having been stained in a deep solution of Prussian blue, the colour being most intense and brilliant; it was not mottled but uniformly stained. The arms and legs showed an icteric tinge; their superficial veins looked as though they had been injected with a solution of Prussian blue and were most beautifully mapped out. On opening the body the chief points noticed were as follows: The stomach contained half a pint of liquid blood; it was deeply coloured blue; it showed softenings and ulcerations in patches, and it was thickened in other parts. The whole of the intestines showed signs of an irritant poison; they were deeply pigmented with the colour and the contents were dark and pitchy. The transverse colon was intensely inflamed and the fat of the great omentum showed bright extravasations of blood and was most striking in appearance. The heart, liver, and kidneys showed signs of commencing fatty degeneration. The lungs and the liver were deeply coloured blue. The brain was rather anæmic, soft, and almost diffluent in parts.

The following case in which a female was tried on a charge of murder and acquitted at the County Louth Assizes on July 3rd, 1896, is of some interest.

The child, a male aged eight months, was seen at 9.30 a.m. apparently in his usual good health. About 2 p.m. on the same day the child was found "dead and cold." At the *post-mortem* examination a lucifer match was found in the œsophagus and five lucifer matches in the stomach. The prosecution gave evidence tending to incriminate the mother and contended that the matches must have been forced down the child's throat. There were some inflamed spots on the stomach, which viscus contained only a very small quantity of semi-liquid stuff, chiefly mucus.

The wood only of the matches remained, the phosphorus and other chemicals having been dissolved, apparently by the liquids of the stomach. It was not probable that dyspnœa, produced by the match in the œsophagus, caused death, as the face showed no lividity and the child had evidently vomited. The cause of death seemed clearly to be the small amount of phosphorus contained on five matches. Free phosphorus was readily detected in the stomach itself. The child appears to have died an hour or two after swallowing the matches. Blyth quotes results of analysis of matches showing that they contain per 100 from 0·012 to 0·055 gramme of phosphorus. The five matches might therefore have contained from only 0·6 milligramme to 2·75 milligrammes of phosphorus.

A case of presumed phosphorus poisoning from an explosive bullet containing phosphorus is reported.[1]

An airman was shot whilst navigating a plane over enemy territory, the bullet entering the left thigh and passing into the abdomen. The wound emitted a visible vapour with the characteristic smell of phosphorus.

The missile fragments were removed by operation but most of the phosphorus estimated at 3¼ grains was not recovered.

For two days the patient made good progress then became semi-comatose. The skin became yellow, anuria developed and death occurred on the sixth day after the injury. The autopsy showed fatty degeneration and necrosis of the liver and fatty degeneration of the kidney.

REFERENCES [2]

Brouardel, " Le Phosphorisme Aigu." *J. de Méd. Int.*, 1902, 6, 171, 184.
Federschmidt, " Fall von Phosphorverg. mit. tötl. Ausgang." *Münch. med. Woch.*, 1906, 53, 2060.
Marer, " Doppelmord mittels Phosphor." *Allg. Wien. Med. Zeitung*, 1899, 44, 443, etc.
Mitchell, " Poisoning by Lucifer Matches." *Glas. Med. Jour.*, 1895, 44 267.
Mowat, " Suicide from Rat Poison." *Lancet*, 1891, 2, 1387.

Chronic Phosphorus Poisoning

Chronic phosphorus poisoning was at one time a serious industrial hazard, but the danger has greatly diminished since the use of yellow phosphorus in match making was discontinued in 1906, by the Berne Convention. Cases may occur in workmen who are exposed to the fumes of phosphorus as for example in the manufacture of fireworks and certain types of ammunition and other processes in which yellow phosphorus is used. Cases have been reported from the inhalation of phosphorated hydrogen (PH_3) in the production of acetylene gas from carbide, and from the escape of this gas from ferro-silicon.

The toxic action is due to exposure to phosphorus fumes which appear to act on the periosteum and subjacent bone causing thickening of both. If there should be damage to the soft tissues enabling the entrance of suppurative bacteria, softening and necrosis of the bone takes place. The poison appears also to have a solvent action on the teeth which leads to caries and this in turn allows access to the periosteum. It can thus be seen that the time factor must be extremely variable since the thickening process may be present for considerable periods before necrosis begins.

The **symptoms** are therefore rarely manifested directly the victim begins to work in a factory, but after weeks, months, or even years, it may be, he begins to complain of nauseous eructations, vomiting, purging,

[1] Bluxland, A. J., *B.M.J.*, 2 : 664 (1942).
[2] *Lancet*, 2 : 241, 1896.

straining, pains in the joints and wasting. The breath and excreta may have a garlic-like odour, the liver may become enlarged and jaundice and anæmia may develop; bronchitis and broncho-pneumonia are occasional complications. Pain in the jaw, swelling and tenderness of the gums, and formation of abscesses discharging fœtid pus through the cheek, roof of mouth, or even the aural cavity, leaving fistulous openings may occur.

For the prevention of industrial phosphorus poisoning, all processes connected with the manufacture must be arranged so that it is impossible for the workmen to inhale the vapour. Although experimentally it has been shown that the inhalation of phosphorus vapours is the cause of chronic phosphorus poisoning, care must be exercised to avoid the ingestion of small quantities from the fingers, etc., and careful and regular examination of the workmen must be made to detect the presence of carious teeth or unhealthy gums, which provide the main portal of entrance. X-ray examination of the jaw should be made if there is any complaint of pain in the jaw, toothache, etc.

Poisoning by Bromine

Source and Method of Occurrence. Free uncombined bromine is practically only found in chemical works and laboratories. There are very few cases of poisoning by it on record.

Toxicity and Fatal Dose. There can be no doubt that bromine is an intense irritant to the stomach or in vapour to the lungs, but no fatal dose can be recorded. Certainly 0·01 per 1,000 in air breathed into the lungs is intensely irritating and injurious, and 0·1 per 1,000 causes actual ulceration of the bronchial mucous membrane. The toxicity of ingested bromine would depend upon the circumstances in which it was taken, having regard to the contents of the stomach.

Symptoms. So far as known, those of a simple but severe irritant (*vide* also pp. 232 and 338).

Treatment. Empty the stomach and treat on general principles.

Post-mortem Appearances. Inflammation of the œsophagus, the stomach and duodenum with some discoloration may be expected and a varying degree of inflammation of the trachea and bronchi with œdema and hæmorrhages. Oedema of the glottis may lead to sudden death.

Analysis. Uncombined bromine may be distilled off by heat from organic admixture; if combined it may be set free by saturating the solution with bichromate of potassium and acidifying with H_2SO_4 before distilling or by acidifying slightly and adding chlorine water. The distilled bromine may be dissolved in chloroform, when it gives a yellow or brown solution. The bromine may be distinguished by its odour, by turning starch paste yellow; by liberating iodine (which gives a blue colour with starch) from potassium iodide; and by giving a white precipitate with solution of phenol. Bromides give a yellowish white precipitate with $AgNO_3$, insoluble in nitric acid.

Poisoning by Chlorine

Source and Method of Occurrence. Free chlorine is found only in laboratories, bleaching-powder factories and other chemical works, and consequently poisoning by it is confined to such places, except for a

solitary case occurring on board a ship carrying casks of chlorinated lime. It was the first poison gas used in warfare, being liberated from cylinders over a 6-kilometre front north of Ypres on April 22nd, 1915. The Allied casualties in this attack were about 5,000.[1]

Symptoms. Exposure to a chlorine concentration of 1 in 1,000 is rapidly fatal; of 1 in 10,000 intolerable for more than a few seconds; of 1 in 20,000 very dangerous if exposure lasts about 30 minutes; of 1 in 100,000 liable to cause bronchitis after 60 minutes or more.

The symptoms in men exposed to toxic concentrations are smarting and running at the eyes, burning in the throat, violent coughing and a feeling of suffocation and pain in the chest. Retching and vomiting with violent headaches lead to great prostration. Dyspnœa is marked, and pulmonary œdema is rapidly developed increasing the distress and leading to marked cyanosis. Sometimes there is circulatory failure with weak, thready pulse and in these cases the prognosis is grave. Occasionally death occurs so rapidly that there is no time for lung changes.

Post-mortem Appearances in Acute Cases. The appearances vary with the time of survival. The essential lesions are inflammation of the respiratory tract, pulmonary œdema, rupture of alveolar walls, hæmorrhages and thrombosis in the lung tissues and increased viscosity of the blood.

In cases in which death is delayed œdema is less, pleural effusion is greater and there is usually secondary infection of the damaged tissues, leading to broncho pneumonia. Occasionally some necrosis of the œsophagus and stomach may be seen, and local irritation of the conjunctiva is to be expected.

Treatment. Consists of complete rest and conservation of warmth, oxygen inhalation and venesection. The latter should be undertaken before pulmonary œdema occurs. Intravenous injection of isotonic salt solution has also been practised.

For references, see " Poison Gases in War," p. 519.

Analysis. Chlorine can be recognised by its smell in a dilution of about 1 per million. It may be absorbed by bubbling the air containing it through a solution of sodium hydroxide, when it forms a mixture of sodium chloride and hypochlorite. This solution, acidified with nitric acid, gives a white precipitate with silver nitrate solution (and other tests for HCl, *q.v.*). Acidified with hydrochloric acid, it liberates iodine from a solution of potassium iodide, and the free iodine dissolves in chloroform to form a violet solution.

A solution of chlorine in water has the same effect on potassium iodide.

Chlorine gives a yellow colour with a dilute solution of o-tolidine in 3·5 per cent. of HCl. This test, capable of detecting 1 mg. of chlorine in a litre of air, can be used for determining the concentration of the gas in contaminated air.

Poisoning by Iodine

Source and Method of Occurrence. Iodine is prepared in large quantities for medicinal and other purposes. It is rarely used as a poison. In 1864 an attempt was made by a woman to poison a fellow-servant by mixing tincture of iodine with food in a plate. The remarkable discoloration of the farinaceous food which it produced led to suspicion,

[1] Vedder, " Chemical Warfare," 1925.

and prevented any ill effects from following. Cases usually occur from overdoses and idiosyncrasy (*vide* "Potassium Iodide," p. 337). When in strong solution, it acts as a corrosive; in this state it has been maliciously employed for throwing on the person.

Toxicity and Fatal Dose. A woman swallowed by mistake one drachm of iodine dissolved in an ounce of alcohol. When seen soon afterwards she complained of a violent pain in the throat or stomach followed by retching and slight vomiting: the pulse was rapid and full, the eyes prominent and suffused. Vomiting, promoted by diluents, brought no relief to the symptoms. She became much depressed, and died on the following day. There was no examination of the body.[1] A case has been reported of recovery after ingesting 6 ounces of tincture of iodine. There was violent abdominal pain and the lips and tongue were severely inflamed and swollen, but the patient was relieved after the stomach was washed out, and made a complete recovery after a few days.

Symptoms. From experiments on animals, as well as from observation of its effects on man, iodine would seem to have a strong local action as an irritant on the stomach and bowels. In large doses, it occasions a burning heat in the throat, severe pain in the abdomen, with vomiting and purging; the vomited matters have the peculiar odour of iodine and are of a yellow colour, except when any farinaceous food has been taken, in which case they are blue or even black. The fæcal matters may also contain free iodine if the poison has been taken in the solid state. Besides these symptoms there is a great thirst, with anxiety, headache, giddiness, trembling, and convulsive movements of the limbs and fainting; these last symptoms indicating that the poison has become absorbed. When taken for some time in small doses, it gives rise to salivation, vomiting, purging, pain in the stomach, and cramps; the pulse becomes small and frequent; there is a general wasting of the body. Iodine produces these secondary effects (iodism), whether it is taken internally or applied externally. For further remarks, *vide* "Potassium Iodide Poisoning," to the iodine of which compound the symptoms are mainly due. The rash, coryza, etc., there mentioned, are sometimes seen when excessive doses of the tincture of iodine have been given.

Treatment. Emetics and the stomach tube may be used, together with general measures to relieve pain, etc., when acute symptoms are present. In other cases, stopping the administration of the drug is all that can be done.

Post-mortem Appearances. As this is an irritant as well as a corrosive poison, the lining membrane of the mouth, gullet, stomach, and intestines may be found inflamed and excoriated. In one instance the mucous membrane near the pylorus was corroded and detached over a space of two or three inches.

Analysis. The odour is characteristic, but it may be concealed by alkalies or alkaline substances. When heated, it distils as a purple vapour, which forms dark violet crystals on cooling. The addition of a cold solution of starch produces a blue colour, but many substances prevent this reaction. It is very soluble in disulphide of carbon, forming a rich pink solution, or in chloroform, producing a violet solution, or in potassium iodide solution, forming a yellow or brown solution. Chloroform or

[1] *Prov. Jour.*, June 30th, 1847, p. 356.

carbon disulphide are suitable for extracting iodine from aqueous solution, gastric contents, tissues, etc. Iodine gives a blue, green, or dark colour to most organic liquids, and imparts to them a peculiar odour. It stains the skin and other organic substances yellow; the colour being removed by an alkali. A solution of iodine in potassium iodide is decolorised by sodium thiosulphate.

Poison Gases in War

The development of these gases for use in war may possibly cause an occasional "incident" in civil life. It is therefore appropriate that some mention of the subject should be made here; but obviously special text-books on the subject must be used for reference.

Such gases may be classified as follows: but it is obvious that many different kinds of gas are probably available although they have so far not been used.

The principal types of gas are as follows:—

I. Acute lung irritants: chlorine, Cl_2; phosgene, $COCl_2$; chloropicrin, CCl_3NO_2, etc.

II. Lachrymators: ethyl-iodoacetate; bromacetone; acrolein, etc.

III. Paralysants: hydrocyanic acid: sulphuretted hydrogen.

IV. Sensory irritants of eyes, nose and chest (sternutators): ethyl-dibromarsine; diphenyl-chlorarsine, etc.

V. Vesicants: dichlordiethyl sulphide, "mustard gas"; B. chlorvinyl dichlorarsine, "Lewisite."

No good purpose can be served by dealing with these substances in detail, and the reader is referred to special works on the subject, such as:—

"The Official History of the War," vol. 2, London, 1923.
Reports of the Chemical Warfare Medical Committee (various).
Chemical Warfare. W.O. Publications.
A.R.P. Handbooks, H.M. Stationery Office.
Winternitz, "The Pathology of War Gas Poisoning," 1920.
Vedder, "The Medical Aspects of Chemical Warfare," 1925.
(This work contains a full bibliography.)
Chemicals in War. Prentiss, A. M. (1937).
Black and Glenny, "Observations on 685 Cases of Poisoning by Noxious Gases used by the Enemy." *J. Roy. Army Med. Corps.*, 1915, 24, 509.

Poisoning by the Chlorinated Hydrocarbons and Similar Compounds

Many such substances, as industrial solvents, cleansing agents, refrigerants, etc., have come into use within recent years. Poisonous effects are most commonly observed in connection with particular employments; but from time to time accidental cases in other circumstances have occurred. A number of suicides from the deliberate ingestion of these substances have been recorded.

Poisoning by Tetrachlorethane

Tetrachlorethane $\begin{matrix} CHCl_2 \\ | \\ CHCl_2 \end{matrix}$ is a colourless liquid boiling at 147° C. and having a vapour density of 1·91 as compared with air. Lehmann[1] showed this to be the most toxic of all the chlorine derivatives of the hydrocarbons. Air containing more than 0·002 per 1,000 produces toxic effects.

[1] *Arch. f. Hyg.*, 1911, 74.

It is known under various proprietary names such as cellon, alanol, novania, westron, etc., and was at one time extensively used in the preparation of the so-called "dope" varnish, which was applied to the wings of aeroplanes so as to form a completely air-tight and watertight coating. A peculiar form of toxic jaundice was observed among those using the varnish, and Willcox[1] established the fact that the poisoning was due to the tetrachlorethane constituent.

It is used in making cinematograph films, in artificial silk manufacture, in making artificial pearls, in dry cleaning, in fire extinguishers, etc.

The affected workers complain of malaise, drowsiness, profuse perspiration, vertigo, insomnia, headache, and constipation.[2] After days, or it may be weeks, jaundice sets in with vomiting, mental confusion and even delirium. The liver, which is at first enlarged, may sustain a marked diminution in weight and size. In fatal cases the liver has been found shrunken, hard and firm and of an intense yellow colour. Microscopically necrosis and fatty degeneration of hepatic and renal cells are marked features. Willcox has emphasised the following clinical features, which may aid the diagnosis : (1) the insidious onset of the symptoms ; (2) the comparatively long duration of the acute stage when marked jaundice has supervened, thus distinguishing the cases from acute yellow atrophy of the liver ; (3) the absence of marked pyrexia, thus distinguishing the cases from infective jaundice ; (4) the absence of anæmia, thus distinguishing the cases from blood poisons ; and (5) the depth of the jaundice.

There would appear to be great variations in the susceptibility of different varnish workers, and much of the poisoning depends on the duration of the exposure and the concentration of the poison in the "dope."

Schwander[3] has reported complete narcosis following absorption of tetrachlorethane through the skin. Forbes[4] describes three cases of poisoning after its ingestion the victims become unconscious and died within 12 hours from central respiratory paralysis. Hunter[5] recognises two groups of cases of chronic poisoning ; in one the predominent symptoms are at first gastro-intestinal with later signs of liver damage ; in the other there is toxic polyneuritis.

Poisoning by Trichlorethylene

Trichlorethylene, alone or mixed with other organic liquids is widely used industrially as a solvent of fats, rubber, etc. It is used also as an insecticide. In high concentration it has a marked narcotic effect and for some years it has been employed as an inhalation anæsthetic less potent but also less toxic than chloroform.

By 1931 it was reported as having caused 284 cases of poisoning in Germany, with 26 deaths.[6] Loss of consciousness occurred in 117 of these cases. Stüber considered that the liver was unaffected, but others have reported jaundice, probably due to trichlorethylene.[7] Dermatitis is sometimes produced by the degreasing effect of the solvent on the skin. Nerve lesions are also described.

[1] Willcox, W. H., *Lancet*, 1 : 544 (1915).
[2] Elkins, Hobby, Fuller, *Jour. Ind. Hyg. Toxicol.*, 19 : 474 (1937).
[3] *Arch. Gewerbepath. v. Gewerbehyg.*, 1936, 1, 109.
[4] *B.M.J.*, 1943, *1*, 343.
[5] *Industrial Toxicology*, Oxford, 1944.
[6] Stüber, *Arch. Gewerbepath. v. Gewerbehyg.*, 1931, *2*, 398.
[7] Willcox, *Proc. Roy. Soc. Med.*, 1933–4, *27*, 455.

Continued exposure to low concentrations produces mild intoxication of a pleasant type such as leads to a craving for repetition.[1]

For *analysis* see section on chloroform.

Poisoning by Methyl Chloride

Methyl chloride, a colourless gas (at room temperatures) with a faint ethereal odour, is used in the dye industry, in the manufacture of chloroform, and as a refrigerant.

Poisoning is usually accidental. In mild cases there is dizziness and headache followed by anorexia, nausea and vomiting. More severe poisoning may incapacitate the victim for several weeks ; ocular symptoms are common (misty vision, diplopia, difficulty in accommodation) ; there is marked weakness and depression. In severe poisoning, besides these symptoms, epileptiform convulsions (sometimes leading to death) may occur ; drowsiness and delirium have been reported ; there is fever ; about half the cases have albuminuria and hæmaturia. Liver degeneration has been found post-mortem, though jaundice has not been reported. Kegel, McNally and Pope[2] give a good clinical description based on 29 cases with 10 deaths. Other cases, among refrigerator repairers, are reported by Jones.[3]

Poisoning by Methyl Bromide

Methyl bromide is a colourless liquid, volatilising at a low temperature to form a colourless gas. Within recent years it has been used extensively in fire-extinguishers, and also as a refrigerating and fumigating agent. From such sources, it has been responsible for a considerable number of cases of accidental poisoning. The systemic effects resemble those described above in connection with methyl chloride. A high concentration of the gas will result in a rapid asphyxial type of death from paralysis of the respiratory centre. Pulmonary inflammation and œdema are common post-mortem findings, and there may be hæmorrhages in the lungs and elsewhere. Degenerative changes in the brain and parenchymatous organs have been described. In addition to its systemic effects, methyl bromide causes burning and vesication of the skin and mucous membranes. There is no specific form of treatment.

Poisoning by Ethylene Dichloride

Ethylene dichloride, a powerful narcotic, was used experimentally as an anæsthetic by Simpson. In industry it is used as a solvent for fats, resins, etc. ; as an insecticide and fungicide : in fire extinguishers, etc.

Death has followed the ingestion of 2 oz. of the liquid.[4] The patient became dizzy, increasingly stuperous and cyanosed with a rapid pulse and death from heart failure occurred in 22 hours. At autopsy the kidneys showed extensive tubular necrosis with calafication as in corrosive sublimate poisoning and the liver showed fatty degeneration.

Inhalation of the vapour has caused dizziness, nausea, epigastric pain and weakness with evidence of liver damage.[5]

[1] Bander, *Z. Gewerbehyg.*, 1927, *4*, 385.
[2] *J. Amer. Med. Assoc.*, 1929, *93*, 353.
[3] *Quart. J. Med.*, 1942, *11*, 29.
[4] Hueper & Smith, *Amer. J. Med. Sci.*, 1935, *189*, 778.
[5] Wirtschafter & Schwartz, *J. Indust. Hyg.*, 1939, *21*, 126.

Poisoning by Ethylene Chlorhydrin

Ethylene chlorhydrin (or Glycol chlorhydrin) is a clear liquid resembling glycerine in appearance and with an odour like that of ethyl alcohol. It is used in the lacquer and paint industries, in dyeing and cleaning, and in the manufacture of linoleum.

Koelsch[1] has reported fatal cases of poisoning with symptoms similar to those produced by other chloro-compounds. Middleton[2] has reported a case in which absorption through the skin seems to have been important, though the vapour was probably inhaled as well. Both authors record œdema of the lungs and broncho-pneumonia.

Poisoning by Diethylene Dioxide ("Dioxan")

Dioxan, used as a fat solvent in degreasing wool, is also employed in the manufacture of lacquers, polishes, cements, cosmetics, etc.; as a paint-remover; and as a fumigant or deodoriser.

Though it is not very volatile, and industrial hazards are slight, accidents have occurred. Barber[3] reported five fatal cases in an artificial silk factory. Premonitory symptoms of nausea, vomiting and abdominal pain were followed by oliguria with albuminuria and hæmaturia. Uræmia supervened and death occurred in about a week. There was no jaundice. At autopsy the kidneys showed hæmorrhagic nephritis with necrosis of the outer part of the cortex and there were areas of necrosis of the liver without bile stain or fatty changes. Similar signs have been recorded by Henry.[4]

Poisoning by Carbon Tetrachloride (CCl_4)

Source and Method of Occurrence. Carbon tetrachloride is a heavy colourless volatile liquid with a boiling point of 76° C. It is non-inflammable. It is in common use as a solvent for fats and oils. At one time it was in common use as a shampoo, but its use has been discontinued owing to its dangerous properties.[5] It is used commercially in making chemical fire extinguishers, in the making of golf balls,[6] in "dry" cleaning (both industrially and in the household), and in certain other trades. It has distinct anæsthetic effects, and has a depressant action on the respiratory and cardiac centres.

Toxicity and Fatal Dose. The substance has long been recognised by chemists as having an anæsthetic action when its vapour is inhaled, and as giving rise to unpleasant symptoms in these circumstances, but no fatal case was recorded previous to July 1909, when the quantity used, and certainly the quantity inhaled, was unknown. The fatal dose must therefore be left as undetermined. Evidence was, however, given to the effect that it was used by the firm in question very frequently without ill effects. Chandler records a case in which severe toxic symptoms arose from inhalation when it was used to remove adhesive tape near the face.[7] The drug has been used as a routine measure in the

[1] *Z. Gewerbehyg.*, 1927, *4*, 312.
[2] *J. Industr. Hyg.*, 1930, *12*, 265.
[3] Guy's Hosp. Rep., 1934, *84*, 267.
[4] Ann. Rep. Chief Inspector of Factories and Workshops, 1933.
[5] See fatal case, *Jour. Amer. Med. Assoc.*, July 31st, 1909.
[6] See case, *B.M.J.*, September 25th, 1920.
[7] Chandler, F. M., *J.A.M.A.*, 107: 2121 (1936).

treatment of ankylostomiasis in doses of 5 c.c. by the mouth. Toxic symptoms sometimes arise, such as headache, nausea, vomiting and convulsions. It is possible that the toxic symptoms are caused by sulphur-containing impurities in the drug.[1] The relatively large doses which may be given by the mouth to dogs[2] without toxic effect are due to the fact that it is not absorbed from the intestinal tract. If alcohol or fat is given at the same time, however, severe toxic symptoms arise. If the vapour is subjected to heat as occurs when it is used as a fire-extinguisher there is a danger of producing phosgene.

In a fatal case privately communicated, a man aged fifty years received a dose of 4 c.c. by the mouth in emulsion of gum arabic. In two hours he felt pain in the abdomen, and was given a purge of magnesium sulphate, and put to bed. Half an hour later the condition became serious, with feeble pulse, dyspnœa, cold sweat and profuse diarrhœa. He remained semi-conscious for two hours, after which complete unconsciousness supervened, and he died in twenty-seven hours from the time when the drug was taken. (As part of the treatment brandy was given, which was likely to increase the absorption of the drug.) *Post-mortem* examination showed necrosis of the liver in larger and smaller patches and fatty degeneration of the kidneys and heart. The large intestine was eroded from chronic dysentery.

Lamson and Wing[3] state that continued doses of carbon tetrachloride produce cirrhosis of the liver.

Symptoms. Faintness and collapse, which rapidly proved fatal, were the only symptoms noticed by the witness of one death. In the non-fatal cases faintness and a sense of suffocation are the symptoms complained of.

Dingley[4] reports a case of poisoning by the inhalation of the fumes of carbon tetrachloride from a fire extinguisher. Some of the fluid splashed on the face of the patient. A burning feeling in the face and eyes was felt, and consciousness was lost in forty-five minutes, the pulse was imperceptible, and there was no apparent breathing. Artificial respiration was carried out for twenty-five minutes, when breathing recommenced. Complete recovery took place.

In less acute cases impairment of renal function is apparently common.[5]

Treatment. Immediate removal into the open air with artificial respiration and inhalation of oxygen must be adopted. Death is apparently very rapid, leaving but little time for treatment.

Post-mortem Appearances. There may be necrotic changes in the liver, and fatty degeneration in the kidneys and heart. There may be irritation of the bronchial mucous membrane.

Analysis. The smell is characteristic. Any of the liquid left can be analysed by ordinary processes. It gives the isonitrile reaction (see chloroform), but does not give vinyl chloride when boiled with alcoholic caustic soda solution.

[1] Khalil, *Lancet*, March 13th, 1926.
[2] Lamson, *Jour. Pharm. and Exp. Therap.*, 1923, 22, 215.
[3] *Jour. Pharmacol. and Exp. Therapeut.*, Baltimore, October 1926.
[4] *Lancet*, May 29th, 1926.
[5] Dudley, *J. Indust. Hyg.*, 1935, *17*, 93.

Group 5. ANAESTHETIC, HYPNOTIC AND ANALGESIC AGENTS

Poisoning by Chloroform
(Trichlormethane, $CHCl_3$)

Source and Method of Occurrence. Chloroform is now prepared in large quantities commercially for anæsthetic purposes, and also for use as a solvent. Questions have arisen from time to time as to the absolute purity of the chloroform used, but so far as we are aware they have not given rise to any medico-legal decisions.

As regards the method of occurrence of chloroform poisoning, probably over 99 per cent. of the cases are due to misadventure in its use as an anæsthetic. Suicides use it very occasionally, and cases from time to time arise in which it is given homicidally and for other criminal purposes, such as robbery and rape.

In cases of alleged robbery and rape, it has been sometimes stated that the person assaulted was rendered suddenly insensible by chloroform ; but chloroform vapour does not produce immediate insensibility. A statement that immediate insensibility was produced strongly suggests that the story is false.

Toxicity and Fatal Dose. *Chloroform Vapour.* The vapour, when respired in a concentrated form, is speedily fatal to life. If it is diluted with a certain proportion of air, it produces insensibility, with entire loss of muscular power in from two to ten minutes, and the patient rapidly recovers after the vapour is withdrawn. The sudden administration of anything over 2 per cent. of chloroform vapour in the air breathed may lead to persistent inhibition of the heart by its action on the vagal centre, or it may lead to fatal fibrillation. At no stage in anæsthesia should the concentration be greater than 0·5 per cent., and usually satisfactory anæsthesia can be maintained by a lower concentration. In experiments on animals the maximum concentration that can be tolerated in prolonged administration is 0·2 per cent.[1]

The intermittent way in which chloroform is frequently given constitutes one of its most potent dangers. It must be administered so that the same level of narcosis is maintained throughout.

In some instances death has taken place within two minutes from the commencement of inhalation. In one of these only thirty drops had been taken in vapour, but the patient died within one minute, and in another, so small a quantity as fifteen to twenty drops proved speedily fatal. Simpson suggested that in some of the alleged fatal cases death may have been attributable to other causes of sudden death.[2]

Its fatal operation is sometimes suddenly manifested after the withdrawal of the vapour. In one case, the heart suddenly ceased to beat four minutes after the vapour had been withdrawn.

The methods by which chloroform when inhaled causes death would seem to be of three distinct kinds.

1. In commencing anæsthesia there may be inhibition of the heart by the rapid absorption of concentrated vapour from the lungs. In other cases in light anæsthesia there may be cardiac fibrillation, especially if there is a sudden alteration in the quantity of the drug inhaled.

[1] Sayers, *et alia*, *Ind. Eng. Chem.*, 26 : 1251 (1934).
[2] *Med. Times and Gaz.*, 1870, 1, p. 224.

2. Poisoning from an overdose during full anæsthesia. This is unlikely to occur if the pulse and respiration are being watched by the anæsthetist. There is probably a toxic action on the medullary centres as well as a direct action on the heart, as shown by perfusion experiments. 3. Delayed chloroform poisoning, by which is meant that after the anæsthetisation and the immediate effects of the operation are both apparently successfully passed through by the patient there suddenly appear symptoms of vomiting, frequent feeble pulse, acetonuria, and apathy deepening into coma. The time in which these symptoms appear is variable ; cases have been recorded in which the symptoms occurred in thirteen, twenty-four, and eighty hours respectively.[1] Fatty degeneration of the heart, liver, and kidneys is commonly found. The ages of the cases mentioned above were 4, 26, and 18 respectively, but from other cases it would seem that children are more liable to be affected. We append the full report of the second case.

The patient, aged 26 years, was admitted with symptoms of ovarian cyst with twisted pedicle. The operation was performed a few hours after admission and consisted in removal of the cyst which was slightly adherent to the intestines. On the following day her progress was satisfactory. She had slept well, and felt well, except for some slight vomiting after the operation. On the following evening—24 hours after the operation—my attention was called to the small quantity of urine passed, only about 16 ounces since the operation ; a catheter was passed, but no urine was found in the bladder. On the following morning the patient was distinctly jaundiced, and the general condition was not good. About 10 a.m. she vomited a large quantity of " coffee-ground " fluid. The stomach was then washed out, and some saline and sodium bicarbonate solution was left in. Her condition seemed to improve. Plenty of fluid was given and was retained ; five grains of calomel were also given. The patient again vomited at about 10 p.m., and from that time onward her condition became progressively worse. There was not much vomiting, but gradually becoming weaker, she died at 2 o'clock on the following morning—60 hours after the operation and 14 hours after the first appearance of the " coffee-ground " vomit, so distinctive a feature of these cases. The temperature had never risen above 99° F., the pulse had gone up to 160 per minute, and the respirations had been normal. The urine, of which about four ounces had been obtained after the symptoms had set in, had given no response to the acetone test.

A limited *post-mortem* examination was made. No peritonitis was found. The specimens, which were examined by Dr. Gouch, showed the following points of interest. (*a*) The liver showed very characteristic changes. The centres of the lobules were quite disorganised, the cells were nearly all necrotic, and much brown pigment was present, and the peripheral parts of the lobules were in a state of advanced fatty degeneration. (*b*) The spleen showed little change except that many of the cells contained brown pigment granules. (*c*) In the pancreas the gland cells showed degenerative changes. No trypsinogen granules were present, but numerous fat globules were found in the protoplasm. (*d*) In the kidneys the glomeruli were normal, and the cells of the convoluted tubules were in some places in a state of advanced fatty degeneration and in others broken down into the lumen. (*e*) In the heart the fibres had almost lost their striation and were filled with fine fat droplets. The condition was not patchy, as in fatty degeneration usually found in anæmic states, but every cell was affected.

When ingested as a liquid chloroform is not a very active poison, a fluid drachm being the smallest recorded fatal dose.

In 1854 a boy, *æt.* 4, swallowed a *drachm* of chloroform, and soon afterwards laid his head on his mother's lap and lost all consciousness. Thursfield saw him about twenty minutes afterwards. He was then insensible, cold, and pulseless. Mustard plasters were applied to the legs ; they acted well, but produced no

[1] *Lancet*, 2, 1909, p. 81.

impression on the sensibility. His breathing varied; it was sometimes natural, at other times stertorous. He became warmer, his pulse full and regular; and he continued *three hours* in this state, when he died quite calmly, without a struggle.

Duration. When chloroform is inhaled with a fatal result, the time that elapses between the first breath of chloroform and death is extraordinarily variable (*vide* above).

When chloroform is swallowed and causes death, the fatal event rarely occurs for some time after the dose has been taken, generally five or six hours. It has been delayed as long as sixty-seven hours (*vide* below, under ("Symptoms").

Recovery is apt to be very slow, though commonly the symptoms (headache and nausea) have passed off in six to eight hours, especially if the patient falls into a natural sleep.

Symptoms. These vary somewhat according to whether the chloroform is inhaled or swallowed.

If Inhaled. The symptoms may be very slight, for after a few breaths the patient may cease to breathe and the heart may stop. More usually, however, there is a stage of excitement followed by one of narcosis, in which heart and respiration cease. Anæsthetists are not agreed as to whether the heart or the breathing stops first, and undoubtedly both must be watched with great care. A universally accepted sign of danger is a *fixed and dilated* pupil. Vomiting is a common result of chloroform inhalation, but not a symptom of great importance *per se*, though it is dangerous on account of the risk of aspirating the vomit.

If Swallowed. Chloroform acts at first as an irritant, and may cause vomiting by direct action on the stomach within a quarter of an hour; should it not do so, however, the symptoms then are sleepiness, rapidly followed by unconsciousness, deepening in fatal cases to coma and death.

A man swallowed *four ounces* of chloroform. He was able to walk for a considerable distance after taking this dose, but he subsequently fell into a state of coma—the pupils were dilated, the breathing was stertorous, the skin cold, the pulse imperceptible, and there were general convulsions. He recovered in five days.[1] A man swallowed nearly two ounces of chloroform. He was seen ten or fifteen minutes afterwards; he had already vomited, and was found insensible with stertorous breathing, and a pulse of about 60. The stomach pump was employed, and some spirit of ammonia was injected. The pulse became more feeble, the breathing slower, and the pupils were insensible to light. The surface was cold, and for a time he continued to get worse, the face becoming purple, while the pulse was intermittent and hardly discernible. Two hours and a half after taking the poison, however, a gradual improvement commenced, but sensibility did not return until four hours later. For several days he continued to suffer from great irritability of the stomach, and eventually he had an attack of jaundice. A man, *æt.* 42, swallowed two ounces of chloroform, and he died in about six hours afterwards. In this case the pupils were fully dilated, the breathing was stertorous, and the skin covered with cold perspiration. He rallied for a short time and then sank again, his lips becoming dark purple and his face livid. On inspection the lungs were found much engorged with blood, and there were some apoplectic effusions in these organs. The stomach was slightly inflamed in patches, and the mucous membrane was softened. It contained a dark fluid, smelling strongly of chloroform.[2] In some cases, alarming symptoms have been produced by much smaller doses, and one of these proved fatal. In March, 1857, a woman swallowed half an ounce of chloroform. Within five minutes she was quite insensible, generally convulsed, the jaws clenched,

[1] *Med. Gaz.*, *vol.* 47, p. 675.
[2] *B.M.J.*, 1866, 1, p. 541, and *Amer. Jour. Med. Sci.*, October 1866, p. 571.

the face slightly flushed, the pulse full and rather oppressed, and she foamed at the mouth. She vomited, and in twenty minutes the convulsions had left her; soon afterwards she had a relapse, and did not recover for twenty-four hours.[1]

A man, *æt.* 58, of robust constitution, but given to drink, swallowed about 1½ fluid ounces of chloroform, with suicidal intent. Six hours later he was found in an unconscious condition. When seen by Brasch his face was flushed, the mucous membranes slightly cyanotic, the eyes closed, the breathing quiet, 20 per minute, but occasionally embarrassed, owing to falling back of the tongue. From time to time he vomited and passed fæces involuntarily. The pulse was small, 80 per minute, the cornea insensitive, the pupils not contracted, and not reacting to light or other stimuli. The patient was absolutely insensible and could not be roused. One-thirteenth of a grain of strychnine was injected subcutaneously, and a quarter of an hour later gr. $\frac{1}{32}$. The pulse became stronger; the patient began to move his hands and arms and to open his eyes for a moment or two; he spoke, though unintelligibly, and vomited mucus mixed with food. The vomited matter did not smell of chloroform. Ten hours after swallowing the poison the man recovered consciousness and complained of thirst, a feeling of internal heat and nausea. The vomiting continued, and next day there was great pain in the region of the liver, which was enlarged and tender. The skin and conjuctiva were jaundiced, the fæces slightly bloodstained. Towards the end there was great difficulty in passing water, and even with the catheter only a few drops of turbid yellowish urine could be drawn off. The patient became gradually weaker, and died, sixty-seven hours after swallowing the poison, of paralysis of the heart and pulmonary œdema. The temperature was normal throughout, the intelligence clear to the last. No *post-mortem* examination seems to have been made.[2]

Treatment. For full details the reader is referred to special works on anæsthetics. If the drug has been swallowed, the stomach must be emptied and well washed out. Subsequently stimulants to the heart and respiration must be administered, and artificial respiration maintained if necessary. Oxygen, strychnine, ether, alcohol, applications of electricity to the phrenics, may all be useful, or whichever of them happens to be available. In the ordinary anæsthetising cases a very important point is to see that the larynx is not obstructed in any way. In vagal inhibition of the heart a dose of atropine may be of value.

Post-mortem Appearances. On opening the cadaver there is very likely to be a smell of chloroform, and when the drug is swallowed the stomach may show signs of irritation in patches; beyond these two signs, which, it must be noted, are neither of them constant, and may both be absent, nothing is to be noticed, and only a critical analysis will reveal the cause of death. There is quite likely to be some evidence of the disease for the operative relief of which the anæsthetic was administered, and this must be carefully noted in case the question may arise as to whether death was due to the disease or operation or to the anæsthetic (*vide* Vol. I). Several cases are reported in which fatty degeneration of organs has been found. This is typically so in the "delayed" cases. A sample of the blood should be collected for analysis in every case.

Analysis. Pure chloroform is a heavy colourless liquid (sp. gr. 1·5), crystallisable at very low temperatures, neutral in its reaction, sinking in water, and only dissolving in that liquid to a slight extent. It has a fragrant odour, and is dissolved by alcohol and ether. A solution in alcohol, in the proportion of one part by volume to nineteen parts of rectified spirit, forms the *Spirit of Chloroform*. Chloroform is highly volatile, but its vapour is not inflammable and not readily combustible.

[1] *Med. Times and Gaz.*, 1857, 2, p. 615.
[2] *Deutsch. Med. Zeitung*, April 7th, 1890; *B.M.J.*, 1890, 1, p. 1089.

It can be decomposed into toxic substances by exposure to high temperatures. Nitric and sulphuric acids produce no change in it. It boils at 142° F., and evolves a vapour which at a red heat yields chlorine and hydrochloric acid. On this effect processes have been based for separating it from the *blood and tissues*, when it has proved fatal in the form of vapour. The substance supposed to contain chloroform is placed in a flask. The neck of the flask is fitted with a stopper perforated to admit a hard glass tube, bent at right angles, and having a length of from twelve to fifteen inches. The flask is gradually plunged into water at about 160° F., and at the same time the middle portion of the tube is heated to full redness by gas jets. At a red heat chloroform vapour is decomposed, and chlorine and hydrochloric acid are among the products of its decomposition. Litmus paper applied to the mouth of the tube is reddened ; starch paper moistened with iodide of potassium is rendered blue, and nitrate of silver gives a white precipitate. Two drops of pure chloroform were thus readily detected, and so persistent was the vapour in the closed vessel, that it was detected after three weeks. Two drops added to a quantity of putrefied blood were detected by a similar process after a fortnight, the flask being closed, but the mouth of the tube remaining exposed to air. This test is not specific for chloroform, but is given by a great many chloro derivatives of aliphatic hydrocarbons. It can thus be used, in conjunction with the history and circumstances, in investigating cases of poisoning by such substances as carbon tetrachloride, tri- and tetrachlorethylene, methylene dichloride, etc.

As chloroform is much less volatile than ether, and its odour is not so pungent, it is not so easily detected in the dead body by the smell. The body should be inspected as soon as possible, and any solids or liquids intended for examination should be kept in well-closed vessels. If the smell can still be perceived in the blood or organs, the vapour may be easily detected by the method above described.

Sir Thomas Stevenson found no difficulty in detecting chloroform in the blood of a person killed by its inhalation, even when the body was not examined till twenty-four hours after death. The flask in the apparatus described above was fitted with a second tube, open at one end to the air, whilst the other end passed beneath the liquid to the bottom of the flask. This served as an inlet for air. The exit tube was heated through a length of at least eight inches ; and its further end was attached to a set of Liebig's bulbs, filled with a strong solution of nitrate of silver. Air was aspirated through the apparatus during the course of the experiment ; and the presence of the slightest trace of chloroform *or other volatile compound of chlorine* was immediately revealed by the formation of a white cloud of chloride of silver in the solution of nitrate of silver.

Pohl[1] describes at length his method of estimating chloroform quantitatively in animal fluids. A current of air is passed through the mixture for many hours ; it takes up the chloroform, and the mixture of air and chloroform being led through a heated tube filled with pure magnesia, the chloroform is decomposed, and can be estimated quantitatively by the amount of chlorine which it yields to the magnesia. Preliminary experiments with known quantities of chloroform convinced him that the method gives almost exact results. The experiments were made on dogs deeply

[1] "Archiv. für exp. Path.," 28, Heft. 3 and 4, 1891.

under the influence of chloroform. In the blood drawn from such an animal there was obtained from 0·01 to 0·06 per cent. chloroform, the average amount being 0·035 per cent., being much less than the solvent power of blood for chloroform. The red corpuscles contained about two and a half times as much as the serum. The greater part of the chloroform is therefore present in the red corpuscles. The amount of chloroform in the brain, liver, urine, and fat relatively to the amount in the blood was also estimated. In a dog in which the blood contained 0·015 per cent., the brain (after death by bleeding) contained 0·0418 per cent. In another dog where the blood contained 0·062 per cent., the liver contained 0·044 per cent. Only traces were found in the urine. The fat contained less chloroform than the blood, but the author attributed this to its very small blood supply. The organs rich in substances which are soluble in chloroform seem to take it up in relatively large amount and quickly. After cessation of administration the blood absorbs it again, and it is excreted by the lungs, the excretion going on for forty minutes at least.

Solutions containing even traces of chloroform evolve the unpleasant odour of phenyl isocyanate when boiled with alcohol and a little caustic potash, on the addition to the boiling liquid of a drop or two of aniline.

Micro-determination of Chloroform in Blood and Tissues.[1] Five cubic centimetres of blood are mixed with six to eight times their volume of alcohol, and acidified with 1 c.c. of a 5 per cent. alcohol solution of tartaric acid. This is distilled and 12 to 15 c.c. of distillate collected in a small flask with a long neck. To this are now added 5 c.c. of a solution of 1 gram Na in 50 c.c. 95 per cent. alcohol, and the mixture is boiled over the water bath under a reflux for about half an hour, which is sufficient to saponify the $CHCl_3$. The following reaction takes place :

$$CHCl_3 + 4NaOH = 3NaCl + NaHCO_2 + 2H_2O.$$

The Cl is then determined. The mixture is first evaporated over the water bath (by plunging the flask into the bath until only part of the neck protrudes outside), and the excess Na alcoholate is decomposed with 1 to 2 c.c. H_2O. When the evaporation is complete, a few drops of HNO_3 and a slight excess of $AgNO_3$ solution (4·267 grams per litre), each cubic centimetre of which corresponds to 1 mgrm. $CHCl_3$, are added. The excess of Ag is titrated with NH_4SCN, 5 c.c. of which equal 1 c.c. of the $AgNO_3$ solution, a micro-burette being used. The absolute error is 0·0066 mgrm. when 1 mgrm. of $CHCl_3$ is used. To carry out this determination on tissues, 5 grams of the material are finely broken up in alcohol, and the determination is continued as already detailed for blood.

The method can be used for other chloro-compounds (*e.g.* CCl_4, C_2HCl_3, $C_2H_2Cl_4$, CH_2Cl_2) which, like chloroform, are decomposed by caustic soda or potash with production of inorganic chloride.

Cases. Some years ago a nurse at the London Hospital, working in the out-patient department, took the chloroform bottle to her bedroom with her and swallowed an unknown quantity. She was found before life was extinct. Her stomach was well washed out, and many other remedies administered, in spite of which she died about twelve hours after taking the drug: on autopsy a curious condition was found which explained the difficulties of treatment. She had an

[1] Maurice Nicloux, *Compt. Rend. Soc. Biol.*, 1924, 91, 1282-1284.

hour-glass stomach from cicatrisation of an old ulcer, and while the proximal end had been washed out, the pyloric compartment smelt strongly of chloroform; in other respects there was nothing unusual in the case.

The following case is interesting from the great prolongation of the period of unconsciousness:

A robust housemaid, aged nineteen, finding herself three months pregnant, took with her to her bedroom on a Friday night a bottle of chloroform and swallowed three fluid ounces of the liquid, most probably between 3 and 4 a.m. on the Saturday morning. At 6.30 a.m. her mistress found her lying with her head over the side of the bed, her cheek resting on the floor. She was snoring loudly and was quite unconscious. Dr. Waterson saw her shortly afterwards, and called in Dr. Robinson at 8.30 a.m. She was in bed, perfectly unconscious, with complete muscular relaxation, flushed face, and widely dilated pupils; the corneae were insensitive. The pulse was regular and full. Her mouth and breath smelt strongly of chloroform. The stomach pump was applied with little result; the fluid which returned did not smell much of chloroform. Strychnine gr. $\frac{1}{30}$ was injected hypodermically, and the dose repeated two or three times during the day. Enemata were given to unload the bowels, so as if possible to get rid of some of the poison; they were not relieved, however, till about 3 p.m. Enemata of strong coffee and of brandy were also given. The mouth gag was used, and the tongue kept well forward all the day. The breathing kept good and the pulse natural. At 4 p.m. consciousness returned after an absence of twelve hours. In the evening she was removed to her home in a cab, and she made an excellent recovery from the chloroform.[1]

In 1886 a woman was tried for the murder of her husband; and it was alleged that she had poisoned him with liquid chloroform poured down his throat whilst he was asleep. She was acquitted.[2]

In April 1904,[3] a man was sent to penal servitude for life for administering chloroform to his two children with intent to murder or do them bodily harm. The accused seemed to have been brooding over some imaginary grievance at the hands of his wife; and early in February he took an unfurnished flat in Maple Road, Camberwell. He bought a quantity of chloroform; took the children to the flat and gave them some of the drug. The next thing they remembered was waking up, feeling very sick and ill, on what they imagined was Monday morning, but, as a matter of fact, was Tuesday; but in fact they had been either asleep or unconscious for two nights and a day. It appeared that a man named Cochrane had lodged with the accused and his wife and that Hallam had been jealous. The whole thing might have been an attempt, on the accused's part, to frighten his wife, or to be revenged upon her for some imaginary slight, but it was clear that, whatever his motive was, he endangered the lives of his children.

The jury, after a brief deliberation, found the accused guilty of administering chloroform with intent to murder, but added that they considered that he was suffering from delusions at the time.

A death in an adult from swallowing an ounce of chloroform is recorded in the *Lancet*, 1897, 2, p. 384.

The following, reported by Dr. Hayward in the *Lancet*, 1902, 2, p. 1122, is inserted for its completeness:

A woman, aged thirty-nine, who had been drinking heavily for some days previously, was found in bed in an unconscious condition between 1 and 2 p.m. Her nightdress and the bedclothes were saturated with an exceedingly offensive blood-stained vomit and faeculent matter, and an empty bottle which had contained two ounces of pure chloroform was found by the side of the bed. A fellow servant had seen her in bed and apparently sleeping naturally at 11 a.m. When seen by me at 2.30 p.m. she was in a deeply comatose state—the face blanched, lips and

[1] *B.M.J.*, 1, 1898, p. 144.
[2] *R.* v. *Adelaide Bartlett*, C. C. C., April 1886.
[3] *R.* v. *Hallam*, C. C. C. 1904.

fingers livid, and the trunk and extremities cold: the pulse imperceptible at the wrist, heart sounds very feeble, breathing shallow but regular, and air entering the lungs. The cornea were insensitive, the pupils equal, semi-dilated, and feebly acting to light. The breath smelt of alcohol, with a suspicion of chloroform, and there was no staining or whitening of the lips or mucous membrane of the mouth.

Having no apparatus at hand, I directed that the bed should be raised, and warm bottles and friction applied to the trunk and limbs, while I left to fetch a stomach tube and hypodermic syringe. On my return, after twenty minutes, I found that the patient had partially recovered consciousness and had vomited a quantity of dark, chocolate-coloured fluid and passed a few ounces of bright blood *per rectum*. The pulse was still imperceptible, but the corneal reflex had returned and the limbs responded to stimulation, while she occasionally muttered a few incoherent words. As she appeared to be recovering consciousness and had vomited freely, it was thought better not to wash out the stomach, which might have entailed risk from heart failure, and the possible chance of fluid entering the lungs. A hypodermic injection of $\frac{1}{10}$ gr. strychnine was given, the foot of the bed raised still higher, and warmth and friction to the extremities continued. As the breathing was stronger and quite regular, it was not considered necessary to employ artificial respiration. The general condition continued to improve, though the heart's action remained excessively feeble. At 4.30 p.m., after she had so far recovered consciousness as to complain of pain in the abdomen, a violent attack of retching occurred; the patient collapsed over the side of the bed, the pupils became widely dilated, the heart ceased beating, and death ensued almost suddenly, the respiration continuing for a few gasps after the heart sounds had ceased to be heard.

A necropsy was made 24 hours after death. *Rigor mortis* well marked. Thorax: the right side of the heart contained dark-coloured blood and no clots; the left side empty, valves normal, walls firm, bright red in colour; weight 13 ounces. Lungs natural on both sides, crepitant, and not œdematous. Abdomen: The peritoneal cavity contained several ounces of dark blood-stained fluid, no lymph on walls of intestines, the small intestine intensely congested and dark red in colour, the large intestine congested to a lesser degree. The stomach contained about one ounce of dark chocolate-coloured fluid smelling of chloroform, the walls slightly congested with a few scattered ecchymoses and patches of *post-mortem* digestion. The interior of the small intestine was intensely congested throughout, the mucous membrane swollen, velvety, and of a dark cherry colour with numerous ecchymoses, and contained a quantity of dark-red fluid smelling strongly of chloroform. These appearances were much less marked in the interior of the large intestine which contained no solid matter. The liver was large, weighing 3½ pounds, pale in colour, somewhat friable. Spleen, kidneys, and pelvic organs normal. The mucous membrane of the œsophagus was very slightly congested and showed no ecchymoses on its walls. Brain firm, normal, the cranial sinuses contained dark fluid blood and no clots. The presence of chloroform in the intestines was confirmed by distillation of the fluid contents. On heating the glass tube leading from the retort, evidence of chlorine gas was obtained by its action on iodised starch paper and on litmus. The general result of the examination pointed to intense irritation and congestion of the alimentary tract, most marked in the duodenum and small intestine, and to a lesser degree in the stomach and large intestine. The condition of the liver may have been due to fatty change, the result of long-standing alcoholism; the cherry-red colour and fluid condition of the blood in the heart and cranial sinuses are also noteworthy.

The following, though not strictly a case of chloroform poisoning, deserves to be recorded as a warning to surgeons:

A man who had received a gunshot wound of the abdomen was brought to the hospital and operated on. The operation was very difficult, and chloroform administration had to be kept up for about four hours. Gas was the illuminant used in the operating room, and it appeared that the gaslight decomposed the chloroform with evolution of powerful chlorinated vapours, which overcame the two surgeons and the Sisters of Mercy. One of the sisters died on the second day, and the lives of the others were in great danger.[1]

[1] *Lancet*, 1898, 1, p. 611.

The following case of **chloroform habit** may perhaps only be rare by reason of its fatal ending.

In October, 1904 an inquest was held at Liverpool on the body of Dr. L. Roberts, who was found dead in an hotel. It was given in evidence that for twenty years he had been in the habit of inhaling chloroform in order to enjoy the beautiful dreams he experienced when under its influence. He would have a bout which would last a few days at a time. His father, relations, and friends did all they could to help him, and tried to persuade him to break off the habit, but he had got beyond that. At times when the bout came on he would absent himself from home for a day or so and even longer. At his death no less than eight chloroform bottles were found in his pockets [evidence does not say empty or full]. Medical evidence showed that the chloroform had been inhaled, not swallowed. A verdict of death from misadventure was returned.

Poisoning by Ether

The action of ether closely resembles that of chloroform. It is considered a safer anæsthetic than chloroform, and certainly fibrillation of the heart and death from vagal stimulation do not occur. It has, however, to be given in greater concentration to produce anæsthesia, and is much more disagreeable and more likely to cause bronchial irritation. About 4 to 5 per cent. of ether in the air is required for anæsthesia. The amount of anæsthetic per 100 c.c. of blood required to cause arrest of respiration is 40 to 70 mgrms. in the case of chloroform and 160 to 170 mgrms. in the case of ether, while the relative amounts to cause anæsthesia are 25 to 35 mgrms. chloroform and 100 to 140 mgrms. ether.[1]

The importance of the control of anæsthesia by supplying a properly regulated concentration of the anæsthetic and increasing the ventilation in the lungs by the supply of a small percentage of carbon dioxide has been insisted upon by Haldane[2] and Henderson.[3]

Ether has been administered by intravenous injection and *per rectum*. It is also commonly taken by the mouth for its narcotic effect, as in the case of alcohol.

Symptoms. When swallowed in moderate doses, it has a hot burning taste, and produces during swallowing a sense of heat and constriction in the throat. It causes great excitement and exhilaration, followed by intoxication, but persons may become habituated to it and thus after a time it may be taken in very large quantities with comparative impunity. Its immediate effects are similar to those of alcohol, but are more rapidly produced, and more transient. Little is known of the effects of ether-drinking upon the duration of life.[4] Ether as a liquid has not often destroyed life. Its symptoms during anæsthesia are similar to those of chloroform.

Analysis. When ether has been taken as a liquid and has caused death, it may be separated from the contents of the stomach by distillation and the product purified by redistillation with dry carbonate of potassium at a temperature of about 120 F. The vapour, bubbled through a dilute solution of potassium dichromate acidified with sulphuric acid (best warmed) will reduce the orange dichromate to green chromium

[1] Cushny, "Pharmacology and Therapeutics."
[2] *Lancet*, July 17th, 1926.
[3] *B.M.J.*, December 19th, 1925.
[4] See *B.M.J.*, 1890, 2, p. 885; 1891, 1, p. 659.

sulphate; and by its peculiar odour ether may be easily distinguished from alcohol or pyroxylic spirit. Ether is highly inflammable, and burns with a yellow smoky flame, producing carbonic acid and water. When it is shaken with an equal bulk of water, only a small portion is dissolved; the rest floats on the surface.

An Unusual Anæsthetic Fatality.[1] A youth, aged sixteen, was being operated on for a fractured jaw, the anæsthetic used being a mixture of ether and oxygen. Warm air was being applied by a dental syringe in order to keep the patient's teeth dry, when an explosion occurred at the back of the boy's throat. Acute hæmorrhage followed, and the boy died within ten minutes.

The syringe was warmed at a flame which was six feet away from the operation table, and there was no naked flame nearby.

Professor Dixon[2] puts the ignition point of ether and oxygen at 220° C., others have placed it as low as 190° C. He points out the difficulty of accurately defining the actual ignition point, but states that there is a special danger in bringing a heated body into contact with such mixtures, even when the heated body is not visibly red hot.

Case. The following case, reported by Dr. J. F. Robertson (*B.M.J.*, 1909, October 30th, p. 1282), is of interest. A man, aged 46, states that he first began to inhale ether as a means of obtaining relief from pains in his chest and body which were the result of indulgence in alcohol, but he only occasionally made use of this means. However, for the last two months he has been more or less constantly inhaling ether, 6 drachms at a time, and getting through a 1-lb. bottle of ether a day, consequently he had been bedridden for weeks, and had lost flesh rapidly and to such an extent that he was unable to stand without help; moreover, the circulation was so feeble that he had hot-water bottles to keep his feet warm. For the last week he had been fed per rectum, partly because he refused to take anything by the mouth, and also because of troublesome hiccough and sickness which followed food given by the mouth. Gradually he became quite apathetic, taking no interest in his surroundings until he reached a stage in which he was quite irresponsible for his words and actions, so that he had to be placed under some restraint and carefully nursed away from his home; I hear he is now progressing satisfactorily, though still very weak mentally and physically. As there was no wasting before he took to bed, and considering the large quantities of ether which were inhaled—no other drug or alcohol being taken—it is reasonable to assume that his present condition is due entirely to the large and continual doses of ether.

During the time he was under my care I never saw any signs of paralysis, nor had he any shooting pains in the legs or other signs of neuritis elsewhere. The patellar reflex was just obtainable, but, as he resented interference, I could not make any detailed examination.

Poisoning by Nitrous Oxide (Laughing Gas)

Source and Method of Occurrence. This gas is now produced in enormous quantities from pure chemical sources for purposes of general anæsthesia. Judging by statistics, it would seem to be the safest of all general anæsthetics.[3] Fatal cases arise practically in only one way, *viz.*, accidentally in the course of surgical operations. The first recorded case was in 1873, since which date they have occurred from time to time—about one in 30,000 administrations.

Toxicity and Fatal Dose. The gas is only slightly poisonous, enormous quantities being sometimes inhaled without any resultant inconvenience.

[1] *B.M.J.*, October 17th, 1925.
[2] *B.M.J.*, January 30th, 1926.
[3] Hewitt, "Anæsthetics."

The fatal dose is unknown, but, as in the cases of other anæsthetics, it probably varies enormously owing to idiosyncrasy of the patient.

Duration. Nothing can be said positively on this point. When it is fatal it usually is so within a few minutes of the inhalation. Insensibility is produced within anything from 20 to 200 seconds, and can be maintained with care for an hour, if necessary, although the gas is usually given only for short operations.

Symptoms. As in other cases of general anæsthesia, the symptoms of inhalation of the gas are, first, a short period of exalted ideation, followed by rapid unconsciousness. When death ensues it is caused by the action of the drug on either the cardiac or respiratory centre.

Poisoning by Ethyl Chloride (C_2H_5Cl)

This drug is mainly used for local anæsthesia by spraying on the part, but is occasionally used as a general anæsthetic. It is very volatile, and therefore the narcosis appears and disappears very rapidly. It has the same toxic effect as chloroform.

Jaeger[1] records a fatal case of ethyl chloride anæsthesia in an alcoholic man, aged forty, after 90 drops, or barely 2·25 c.c., of the anæsthetic had been given. The operation was an exploratory excision of an ulcer of the leg suspected to be carcinomatous. The autopsy showed fatty degeneration of the heart, a small area of consolidation in the upper lobe of the left lung, and calcification of the retroperitoneal and prevertebral glands. The case shows that in employing ethyl chloride anæsthesia special attention should be paid to the condition of the heart, and the mere suspicion of degenerative changes should serve as a contra-indication to its use. For this reason the greatest care should be taken in administering ethyl chloride to alcoholics. When the heart is not absolutely sound ether anæsthesia is preferable.[2]

Analysis. Ethyl chloride is a colourless liquid with an ethereal odour and a burning taste. It boils at 12 –13° C. It burns with a smoky flame, and the combustion products include hydrochloric acid and chlorine (*cf.* under Chloroform).

Poisoning by Methylene Dichloride (Dichlormethane CH_2Cl_2)

Source and Method of Occurrence. Methylene dichloride is a highly volatile liquid and has been used as a substitute for the vapour of chloroform in surgical operations. It was thought to be less likely to cause death. Like all anæsthetic vapours, it has, however, destroyed life on several occasions, even when given with care. The history of these fatal cases is similar to that which chloroform vapour has furnished on numerous occasions.

Symptoms and Appearances. An operation for artificial pupil was about to be performed on a man, *æt.* 40. Five minutes after the vapour of methylene dichloride was given, when the operation had just commenced, the face of the man became livid, the breathing difficult, and the heart suddenly ceased to beat. On inspection, the principal appearance was congestion of the lungs. In another case a man inhaled, for

[1] *Zentralbl. f. Chir.*, July 30th, 1921.
[2] *B.M.J.*, Epit., October 8th, 1921.

the purpose of a trivial operation, a drachm and a half of the vapour. It was given in the usual way by an experienced person, and was stated to be not more than one-half of the usual dose. The deceased became insensible—the operation was completed in a minute—when it was noticed that the patient's head had fallen on one side, his eyes were upturned and breathing and pulsation had ceased. Animation could not be restored. On inspection all the organs of the body were found healthy. Nothing was found to account for the death except the anæsthetic. Methylene dichloride has sometimes been used under the name of chloroform. In 1869, a man to whom the vapour was administered at Charing Cross Hospital died within two minutes from the effects, although it was administered with care, and by one experienced in the use of chloroform. The allegation, therefore, that this vapour possesses any greater degree of safety than chloroform in surgical practice is not supported by facts.

Analysis. The liquid has a peculiar odour, resembling that of chloroform. It is not inflammable, but burns in contact with flame with a smoky combustion. It is not very soluble in water, but sinks in it, the globules having an opaque appearance. It has no acid reaction. Nitrate of silver gives no precipitate with it. If it is boiled under a reflux condenser with an alcoholic solution of sodium hydroxide it yields, *inter alia*, vinyl chloride (recognised by its odour of garlic) and hydrochloric acid. Chloroform, under these conditions, gives no vinyl chloride. On the other hand, methylene dichloride does not give the isonitrile reaction given by chloroform.

A mixture of chloroform and ether has been sold as bichloride of methylene.

REFERENCES

Buchan, "Death from Administration of Methylene." *Lancet*, 1884, 2, 91.
Chamberlayne, "Death under Methylene." *Lancet*, 1891, 2, 486.
Hughes, "Death during Administration of Methylene. Necropsy." *Lancet*, 1890, 2, 768.

Poisoning by Alcohol

Ethyl Alcohol (C_2H_5OH).

Ethyl alcohol is produced by the fermenting action of yeast on sugars, and is concentrated by distillation. The percentage of absolute alcohol in various beverages is approximately as under, taking round figures without regard for precise accuracy :

Spirits, such as whisky, brandy, gin and rum, contain about 40 per cent. of alcohol.

Fortified wines, such as port and sherry, contain about 20 per cent. of alcohol.

Natural wines, such as claret and burgundy, contain about 10 per cent. alcohol.

Strong beer contains about 5 per cent. of alcohol.

A "large whisky," which should be half a gill ($2\frac{1}{2}$ ounces), contains therefore 1 ounce, or 30 c.cm., of absolute alcohol, and the same amount will be present in a pint of strong ale, half a pint of claret, or a quarter of a pint of port.

The absorption of alcohol from the stomach and small intestine begins soon after ingestion. The rate of absorption is dependent upon a number of factors, the most important being the presence or absence of food in the stomach. Food delays absorption and the delay is most marked in the presence of fat and protein (Haggard and Greenberg, 1934 ; Mellanby,

[1] *Pharm. Jour.*, 1871, p. 875.

1919[1]). The concentration of alcohol is important, and generally the stronger the drink the more rapid is its effect. Absorption is usually complete within the first hour, so that after a single dose the maximum concentration in the blood is reached within the same period. After absorption, the alcohol is distributed more or less evenly throughout the tissues, with the exception of the bones and fat. Thus, by estimating the amount of alcohol in the blood, it is possible to calculate the approximate total quantity in the body at that time and the minimum quantity which must have been ingested. About 90 per cent. of the alcohol absorbed is oxidised and the remaining 10 per cent. is excreted, mainly by the kidneys and the lungs. At no stage in its oxidation is alcohol stored in the tissues and its disappearance from the blood takes place at a fairly uniform rate, which for rough calculation may be placed at 10 c.cm. per hour (0·185 c.cm. per kg. of body weight (Mellanby, 1919)[11]). It thus takes about three hours for the blood to be cleared of alcohol after the ingestion of a single large whisky. The excretion by the kidney is of importance, in that at the time of secretion the urine has a similar concentration as that in the plasma at the same time, though higher than that in the blood as a whole, the ratio being approximately 1·3 : 1.[2] The concentration in the blood varies, however, increasing during absorption and then decreasing with oxidation. It follows, then, that the concentration of alcohol in a given sample of urine will correspond with the average concentration in the blood during the time the urine has been collecting in the bladder (Smith and Stewart, 1932)[3] If the concentrations in the urine and blood are estimated some time after ingestion, when the alcohol in the blood has fallen through oxidation, it may be found that the urine alcohol is at a higher level. Urine examination may therefore be used instead of blood examination and gives a reasonably accurate idea of the total alcohol in the body.

A considerable number of investigations have been made in an endeavour to correlate the alcoholic concentration in the blood with the behaviour of the individual.

It is generally agreed that with concentrations below 0·05 per cent., there is little change to be observed on clinical examination; at 0·10 per cent. a number show mild symptoms and quite possibly some more decided symptoms. Between this level and 0·2 per cent. the number showing decided symptoms of intoxication increases, and at the latter figure it is to be expected that practically all will be diagnosed clinically. The critical concentration seems to lie at or about the 0·15 level and any person with this amount in his blood can be considered to have imbibed a dangerous amount of alcohol. With increasing concentrations the symptons become more intense and at concentrations beyond 0·2 per cent. up to 0·5 per cent. there is likely to be marked inco-ordination, coma and possibly death.

Knowing that the concentration in the blood is the same as that in the tissues, we may translate these percentages into amounts drunk as follows. When the amount in the blood is equal to 0·15 per cent., the person has ingested as a minimum within a few hours of the test 1·5 c.c.

[1] Mellanby, Med. Research Comm. Report, 31, 1919; Miles, *Jour. Pharm. and Exp. Therap.*, 1922–23, 20, 265; Haggard & Greenberg, *J. Pharmacol.*, 1934, 52, 150.
[2] *Biochem. Jour.*, 1925, vol. 19, No. 5, 737.
[3] *B.M.J.*, 1932, 1, 87.

of absolute alcohol per kilogramme of body weight. In a man of ten stones this would amount to about $3\frac{1}{2}$ ounces of absolute alcohol, or about $1\frac{1}{2}$ gills of whisky at proof.[1]

Effects of Alcohol. The only acute effect of alcohol which is of any interest is its effect on the central nervous system. Its first effect appears to be a depression of the highest evolutionary centres, the centres regulating the conduct, judgment, and self-criticism. It passes progressively downwards through the centres of earlier evolutionary origin until the motor centres are reached, and finally it depresses and paralyses the vital centres in the medulla.

There is first a feeling of well-being and a certain slight excitation. The actions, speech and emotions are less restrained, due to a lowering of the inhibition normally exercised by the higher centres of the brain. With this there is increased confidence and a certain carelessness of consequences. **This implies a lack of self-control, which is one of the first things observed after alcohol, and which is a constant feature of alcoholic poisoning.**

When the narcosis has penetrated more deeply the sense perceptions and skilled movements are affected. The increased loss of the inhibitory action of the higher centres causes an alteration in the conduct of the individual according to the dictates of his inherent desires and emotions.

This accounts for the fact that an individual may become morose, gay, irritable, excitable, pugnacious, sleepy, and so on, according to the dominant impulses which have been unleashed by the drug. The reaction times are somewhat lengthened, and there is a certain clumsiness and inco-ordination in the finer and more skilled movements shown by slight alteration in speech and in the finer finger movements.

This passes into a third stage, where the motor and sensory cells are deeply affected; speech becomes thick and slurring; co-ordination is markedly affected, causing the patient to stagger and possibly to fall. Finally a stage is reached where the narcosis affects the whole nervous system, and the patient passes into a state of coma with stertorous breathing, indicating a commencing paralysis of the respiratory centre.

The coma gradually lightens into a deep sleep, and the patient, if left alone, usually recovers in eight to ten hours, and wakes up with gastro-intestinal irritation, and usually nausea, vomiting and severe headache. If the coma continues for more than ten hours, the prognosis is bad.

Tests. - A great deal of work has been done to ascertain if there is a measurable effect of alcohol in its earlier phases on the various reflexes and on co-ordinated actions which might be made use of in diagnosis. A list of these works appears at the end of this article, from which the reader may obtain such information as there is. The tests vary from such simple reactions as the speed of reaction to sight and sound and movements of the eye, to more complicated tests involving judgment and discrimination.

It may be accepted from a careful perusal of the available literature that there is in general a lengthening of reaction time and diminution of discrimination with moderately small doses of alcohol, the magnitude of the delay in reaction time depending upon the degree of complication of the task to be done. Many of the tests were carried out after a dose of

[1] "Alcohol: its Action on the Human Organism," H.M. Stationery Office, 1924.

alcohol equivalent to one large whisky, producing a concentration of approximately 0·05 per cent. in the blood. The slowing of the reactions can be shown before there is any clinical manifestation of intoxication. The lengthening of reaction time varies considerably and is often not of great extent, but it must be remembered that a delay of 1/10 of a second means that a car travelling at 50 miles per hour needs an additional 7·3 feet of road in which to pull up.

In practice, several tests have to be made, for from time to time an individual is examined who improves under the influence of alcohol in one or other of the tests ; although it is agreed that if a sufficient number is applied, no subject ever improves in all.

Tolerance is exhibited to a considerable extent, and the reason for this tolerance is not certain. Some observers have stated that in the seasoned toper and in the abstainer the same percentage in the blood produces similar effects, and that in the case of the toper the absorption is slower, that the drug does not reach the same percentage with equal amounts ingested, and that it disappears from the blood more rapidly. There is not yet sufficient evidence to be certain about these points, and Prigsheim[1] has proved experimentally that a tolerant animal shows no signs of intoxication when its blood contains enough alcohol to produce definite toxic symptoms in an ordinary animal. It is certain, however, that a person in the habit of taking alcohol daily can drink alcohol, without being " drunk ", in quantities which would gravely affect a person unaccustomed to the drug.

In certain cases, especially after injuries to the head, after an attack of insanity or of delirium tremens, there may be a greatly increased susceptibility to alcohol.

Symptoms and Diagnosis of Acute Drunkenness. From the above short description of the effects of alcohol, it is obvious that there is no one sign which is specific to this drug, and a diagnosis can be arrived at only by a careful consideration of the whole of the symptoms and signs.

In the early stages where there is a simple loss of control, no precise diagnosis is possible, for to all intents and purposes the person might be a normal individual. It is quite possible that any one in daily contact with the individual could tell at once that his conduct was abnormal, but this is beyond the reach of the ordinary observer, and the special tests for perception, discrimination and control, give no real help in arriving at a diagnosis.

In a more advanced stage there may be obvious signs of alcoholism. The breath smells of drink. The smell is not purely alcoholic, but usually has an unpleasant component, especially if the drink has been taken over a long period. The face may be flushed, the pupils slightly dilated but reactive to light, and the conjunctiva congested. The pulse tends to be accelerated and of low tension. The surface temperature is usually raised, the internal temperature sometimes reduced.

There is difficulty in fixation of the eye, and convergence is limited. Diplopia may occur. Indistinctness in speech may be noted, varying with the stage of intoxication and with the individual. The repetition of difficult phrases may have a certain value in estimating the loss of co-ordination of speech. Hiccup may be present.

[1] *Biochem. Zeitsch.*, 1908, 12, 143.

There may be a certain inco-ordination of the upper or lower limbs, which should be tested in turn. Disorder of the higher centres may be tested by noting the demeanour of the individual, whether he is excited, talkative, abusive, etc. His memory for recent events should be tested by asking him to describe the events leading up to his arrest and subsequently. His knowledge of the passage of time should also be tested, for this is affected fairly early. There is no specific effect on the reflexes commonly observed in practice.

The various pathological or physical conditions which may give rise to the symptoms enunciated must be excluded, and in this connection, it is always advisable to make a further examination some hours later, or the " morning after." The examination of the urine quantitatively for alcohol may be of considerable value in arriving at a conclusion as to whether the person is intoxicated by alcohol.[1]

Having finished the examination, the question must arise whether the person is " drunk " or not for the purposes of the courts. It must be remembered that drunkenness by itself is not an offence unless it is accompanied by some act or omission which causes danger to the life or property of the individual or to some other persons. Thus a person is not charged simply with being drunk, but with being drunk and disorderly, drunk and incapable, drunk in charge of a vehicle, etc., etc.

The effect of the alcohol on the conduct of the individual is therefore the important issue, and the point at which the influence exerted by alcohol may cause danger to the person or to others varies with the particular occupation of the person at the time of arrest.

The term " drunk " is incapable of receiving a general definition, and by it we mean that a person was so affected by the action of alcohol that his conduct was sufficiently abnormal to lead to his arrest in the particular circumstances charged. This definition obviously includes vastly different stages in the intoxication by alcohol, for a man driving a motor-car in traffic might be considered drunk, whereas a person under the influence of alcohol to the same extent, but driving a horse vehicle, might not be considered drunk, and further, the stage at which the horse-driver might be considered drunk would not necessarily be held reprehensible in the case of a pedestrian.

In giving evidence about the condition of such a person, the practitioner should not use the term " drunk " if it can be avoided, but should state whether the person had taken alcohol or not, *and whether in his opinion the alcohol was taken in sufficient quantity to affect the patient to a dangerous extent in carrying out the occupation in which he was engaged at the material time.* In the deeper stages of intoxication the diagnosis is not difficult and can usually be made safely by the average policeman.

The Road Traffic Act, 1930, Section 15, runs as follows :

> " Any person who when driving or attempting to drive, or when in charge of a motor vehicle on a road or other public place is under the influence of drink or a drug to such an extent as to be incapable of having proper control of the vehicle shall be liable . . . "

[1] Mr. Justice Rigby Swift, Liverpool Assizes, February 14th, 1934, stated that the police (a police surgeon) have no right to apply tests for drunkenness without the consent of the accused, and a similar ruling has recently been given by a full bench of Scottish judges in making their decisions in a case of appeal.

Though the wording of the Act widens the scope of a possible charge by including conditions which a layman would not ordinarily describe as drunkenness, it in no way lessens the difficulty of deciding what degree of influence in intoxication by alcohol or other drug can be regarded as constituting a standard beyond which a person can be said to have rendered himself incapable of controlling a motor vehicle.

When a practitioner is summoned to examine such a suspect, it is his duty to inform him who he is and why he has been asked to examine him, and in the presence of witnesses ask his consent to proceed with the examination, informing him of his right to refuse, and that if he consents the results of the examination will be recorded and used either in his favour or against him as the case may be. It has been argued that if a man is so much under the influence of alcohol as to be certified unfit to drive a car he is not in a fit condition to give or withhold consent, and that an examination carried out in these circumstances is illegal. Most of these cases, however, claim that they are not drunk and are perfectly fit to drive, so that the aforementioned argument is rather two-edged, for by claiming at trial that the examination is illegal they admit the justice of the charge preferred against them. A further legal point should be noted, namely, that if the examining Surgeon certifies that the suspect in his opinion is too much under the influence of alcohol to be in charge of a car, he must inform him of the fact and of his right, if dissatisfied with the finding, to have a doctor of his own choice examine him at his own expense.

It has already been stated that there is no legal definition of the word "drunk," but it is desirable that the examining doctor should have in his mind some sort of standard by which to formulate his opinion. In 1927 a Committee of the British Medical Association, specially appointed for the purpose, produced a report on *Tests for Drunkenness;* from their deliberations the Committee came to certain conclusions which are quoted here *in extenso* :—

Classification of Tests

The Committee having carefully considered the tests at present in use for establishing the fact that an individual is under the influence of alcohol, also those brought to notice by recent scientific research, has come to the conclusion that tests should be applied with a view to determining the following facts :—

(*a*) Whether the person concerned has recently consumed alcohol.

(*b*) Whether the person concerned is so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time.

(*c*) Whether his state is due, wholly or partially, to a pathological condition which causes symptoms similar to those of alcoholic intoxication, irrespective of the amount of alcohol consumed.

The tests were consequently considered under each of these headings and conclusions arrived at as follows :—

1. Tests to Prove that a Person has Recently Consumed Alcohol

(i) For the purpose of determining whether or not a person has consumed alcoholic liquor there is no test of such ready application and practical value as "smell."

(ii) The smell of alcoholic liquor in the breath or in vomited matter (if any) is a sign of the consumption of alcohol.

(iii) A factor which must be borne in mind in regard to the smell of alcoholic liquor in the breath is that the intensity of the smell varies according to the nature of the liquor that has been consumed and to the time that has elapsed since its consumption.

(iv) If there is no smell of alcoholic liquor in the breath of a person seen within a reasonable time after his arrest, it is improbable that he has recently consumed alcohol, and search would be made for some other cause of the condition concerning which an accusation has been made against him.

(v) Keenness of the sense of smell varies in different individuals who may be applying this test. This fact must be taken into consideration, and also the fact that the smell of alcoholic liquor in the breath may be disguised by various substances.

2. Tests to Determine Impairment of Control of Faculties

(i) There are no tests universally applicable for determining the amount of alcohol which would render a person incapable of carrying on his occupation in a proper manner, as the effect of alcohol varies within wide limits in different individuals and in the same individual under differing conditions. Fine shades of self-control might be lost without any apparent signs of alcoholic intoxication.

(ii) The first effect of alcohol is on the higher centres and is subjective, and even if no objective symptoms occur the subjective effect of alcohol may be sufficient to make it unsafe for an individual to be in a responsible position, for example, in charge of a mechanically propelled vehicle.

(iii) There is *No Single test* which *Taken by itself* would justify a medical practitioner in deciding that the amount of alcohol consumed had caused a person to lose control of his faculties to such an extent as to render him unable to execute safely the occupation on which he was engaged at the material time. A correct conclusion can only be arrived at by the consideration of a combination of several tests and observations such as :—

General demeanour ;
State of the clothing ;
Appearance of conjunctivæ ;
State of the tongue ;
Smell of the breath ;
Character of the speech ;
Manner of walking, turning sharply, sitting down and rising, picking up a pencil or coin from the floor ;
Memory of incidents within the previous few hours, and estimation of their time intervals ;
Reaction of the pupils ;
Character of the breathing, especially in regard to hiccup.

(iv) The following are tests upon which, taken by themselves, little stress should be laid in deciding whether or not a person is under the influence of alcohol :—

Presence of tachycardia (rapid pulse) ;
Repetition of set words or phrases ;
Character of handwriting ;
Walking along a straight line ;
Failure of convergence of the eyes.

3. Tests for Pathological Conditions Simulating Alcoholic Intoxication

(i) The pathological conditions which may cause symptoms resembling those of alcoholic intoxication, irrespective of whether or not any alcoholic liquor has been consumed, come under certain of the following groups or diseases :—

A. Severe Fevers.
B. Acute Inflammatory Lesions of the Brain or the Cerebral Meninges.
C. Other less Acute Lesions of the Central Nervous System.
D. Mental and Nervous Disorders.
E. Diseases in which General Metabolism is probably at fault.
F. The Results of Head Injuries.
G. Vascular Lesions of the Brain.
H. The Acute Effects of Drugs.
I. The Chronic Effects of Chemicals.
J. The Effects of Extremes of Temperature.
K. Excessive Loss of Blood.
L. Stokes Adams Disease.
M. Sudden Nervous Shock.
N. Hysterical Trance.
O. Auto-Intoxication.
P. Acidosis.

(ii) Diagnosis would have to be decided (*a*) by a chemical examination of the urine, including tests for pathological and toxicological abnormalities, and of the vomit (if any) ; (*b*) by an examination of the knee-jerks and other reflexes, the pupils, retinæ, heart, and organs of respiration ; and (*c*) by an examination for signs of kidney disease and for signs of intracranial disease, such as retraction of the head and hemiplegia or any paralysis of the cranial nerves.

(iii) *There is no single symptom due to the consumption of alcoholic liquor which may not also be a sign of some other pathological condition.* The value of these points detailed above as established by practical examination may now be considered.

General Demeanour. The suspect alcoholic may exhibit in his general behaviour any of the emotions to which human beings are liable, and almost every such mood has been seen in the examination of such cases. Probably the individual shows an exaggerated condition of what is his own prevailing mood. The loquacious mood, with a marked tendency to repetition of some phrase or sentence is amongst the commonest seen. Occasionally, conversely, one encounters the silent, dour type, or again the aggressive, the abusive, the suspicious, the sentimental and emotional

element may be prominent, and frequently, too, a completely irresponsible attitude to the seriousness of his position may be shown, or more rarely the reverse state of remorse and deep concern. It is unusual not to be able to note some departure from what one usually considers the normal.

State of the Clothing. The clothing may be soiled in a way one does not expect in one of the social class to which the accused happens to belong —the accused may be of any class— or the clothing may be improperly adjusted, *e.g.*, an open fly with complete unconcern on the man's part. In the great majority of cases, however, the clothing is quite correct.

Appearance of the Conjunctivæ. These are invariably injected in certified cases, but one must remember similar effects especially on motor cyclists from exposure to cold and dust.

State of the Tongue. By far the commonest sign here is a dry, furred tongue—excessive salivation, sometimes described, is very rare. It is extraordinary how rapidly this dry, furred condition forms and a similar dry, glazed appearance of the fauces frequently accompanies it.

Smell of the Breath. There is nothing to add to the statements in Section 1 above of the British Medical Association report.

Character of the Speech. Thickness of speech, slurring of consonants, and general difficulty of articulation are very common. These are better elicited by asking the suspect his name and address and occupation, or by getting him to read a paragraph from a newspaper, than by asking him to repeat any of the stock phrases or words frequently employed, over which many a normal individual may stumble. Particular care is necessary to make sure there is no physical defect to account for the articulation defect.

Manner of Walking, etc. This often shows significant features—either in swaying in ordinary walking or staggering in turning, or by the individual walking with a broad base and requiring space in which to turn. At other times the obviously deliberate type of walking in an effort to do well marks a departure from the normal. Walking a line and heel and toe walking are difficult to some normals. Walking on stairs also provides evidence at times—the walk upstairs often being accomplished quite well, but in descent when the man has to preserve his balance while the knees are bent he reveals definite inco-ordination in his movements. Fumbling movements in picking up a pencil from the floor may at times also be seen, but are not so frequently in evidence.

Memory of Incidents within the Previous Few Hours and Estimation of Their Time Intervals. This is one of the most valuable tests. Most people can at any time of the waking day tell the time at least within an hour, but in those under the influence of alcohol, even when inco-ordination signs are not prominent, there is frequently marked memory defect in this respect, errors of three or four hours in the estimate of the time being frequent. These nearly always err in the direction of giving a time earlier than the actual time, and frequently correspond to the time at which the individual commenced to imbibe, *e.g.*, if at midnight a man states he thinks it is 8 o'clock, it will probably be found on further investigation that this was the hour when his drinking started and the intervening hours have been "concertinaed" in his memory. Not infrequently too when this sign

is present it will be found that the man gives two quite divergent answers if asked the same question at the beginning and towards the end of his examination. It is also helpful to try to get an accurate account of the happenings of the two or three hours immediately preceding the examination—as this also reveals gross memory defects at times.

Reaction of the Pupils. The evidence from examination of the pupils ranks equally with the previous sign in value. Almost invariably in cases certified it has been found that the pupil is widely or moderately dilated and that the reflex for light is either absent or very sluggish, even when carried out in the dark with the aid of the light from a strong torch.

Character of the Breathing and Presence of Hiccup. When the breathing is affected and hiccup has developed, other signs are usually strongly positive.

Of the tests on which the Committee state little stress should be laid by themselves, mention has been made of *repetition of set words or phrases*, and of *walking a line*. *Failure of convergence of the eyes* has not been found helpful. *Tachycardia*, however, is invariably present in those under the influence of alcohol, and though sheer excitement or nervousness may produce the same symptoms, its absence is definitely in favour of the accused.

Character of Handwriting. This is a useful test. It is true that excitement may cause irregular handwriting, but the gross nature of the irregularity so frequently seen is not likely to be accounted for in this way. But there is something more significant. If a man is asked to write his name and address, he is asked to do something which for most people is almost an automatic action, but in those under the influence of alcohol it is very frequent to find in their signatures and addresses, little errors of spelling which one would not expect and which indicate definite lack of co-ordination between the hand and brain. These errors take the form usually of letter duplication, or of duplicating the wrong letter, e.g., Aberdeen may be spelt ABERDENN, or ABERDDEN, or Great spelled GREEAT or Street, STRRET or STRETT. These small errors may be of considerable importance in estimating inco-ordination.

In the British Medical Association report it is stated that "there is no single symptom due to the consumption of alcoholic liquor which may not also be a sign of some other pathological condition" and a long list of conditions is given which have to be borne in mind. Malaria is occasionally and unexpectedly encountered, and Locomotor Ataxia, Spastic Paraplegia and G.P.I. have all been seen. Head injuries are common and may cause confusing symptoms, so much so that if a serious head injury is present it is unwise to certify that symptoms resembling alcoholism are in fact due to alcohol.

The effects of various drugs may be very similar to the effects of alcohol, *e.g.*, the effects of Belladonna with the dilated pupils, blurred vision, dry tongue, staggering gait, difficulty in speech and excitement, have at times caused confusion; and luminal may also cause doubt at times. In diabetics an overdose of insulin producing hypoglycæmia results in a state almost indistinguishable clinically from drunkenness, and an Airdrie bus driver was convicted under Section 15 of the Road Traffic Act (which includes not only the effects of alcohol but "of a drug") because of inability to drive from hyperinsulism. The effects of intense cold have

also been encountered as a confusing element. The effects of carbon monoxide in a badly ventilated car have also been claimed to cause confusing symptoms and it is undoubted that carbon monoxide fumes may produce mental confusion, staggering gait and inco-ordination. This does not occur till there is a blood concentration of about 30 per cent., so that to substantiate such a defence it would have to be shown that either the exhaust defect was gross, or that the exposure was prolonged. A spectroscopic examination of the blood if carried out at the time would of course prove or disprove the case. These and all other possible conditions which may increase the difficulty of diagnosis can only be eliminated by great care in clinical diagnosis and by bearing all such possibilities in mind at the time of examination.

As previously mentioned, there is a relationship between the alcohol present in the urine and the amount circulating in the blood. It is advisable, therefore, to take a sample of urine from the patient if he does not object, and another sample after a quarter to half an hour. The second sample when quantitatively analysed gives information as to the concentration of alcohol in the blood at the time of arrest. If a sample of blood can be obtained for analysis so much the better. We are not yet in a position to state that a certain concentration in the blood connotes a certain stage of intoxication, but the knowledge is of value in supporting clinical evidence and does give definite information about the amount ingested. In many of the States in America precise limits have been laid down, and if a person is found to have that percentage of alcohol in his blood it is in itself accepted as proof of intoxication. The reader will remember that one large whisky in a medium-sized man produces a concentration of approximately 0·05 per cent. in the blood. The most commonly accepted figure indicating intoxication is 0·15 per cent., which represents a dose of three large whiskies. It is generally known that cases occur in which one whisky produces definite intoxication, and all are familiar with others in which three large whiskies may produce little apparent effect, but the question arises, even if there is no precise clinical effect, whether the effect of this amount of alcohol on the reactions is likely to make a man a bad driver. None of the tests proves this, but they all show that there is a diminution in those reactions and controls which might diminish the driver's skill. It would appear also that alcohol tends to cause a driver to increase his speed without knowing it, and to take risks that he would not ordinarily take. It is equally well known that some drivers, knowing the effect of alcohol upon them, habitually exercise more care than usual after drinking.

An interesting series of observations made in the United States (Holcomb, 1938) gives further light on this question. In one investigation of cases of personal accident when the drivers could be examined, it was found that 47 per cent. of drivers involved in accidents had been drinking. Twenty-five per cent. of them (*i.e.*, more than half of the drivers who had been drinking) had 0·1 per cent. of alcohol in the blood, while 14 per cent. (*i.e.*, nearly a third of the drivers who had been drinking) had as much as 0·15 per cent. or over. Holcomb made a further investigation of a general cross-section of drivers in a certain locality over a period of a week. These consisted of 1,750 drivers examined on the road at different periods of each day and night. He ascertained that 12 per cent. of these drivers had been drinking and as they comprised a fair cross-section

it may be assumed that about 12 per cent. of all drivers are in the habit of drinking to a greater or less extent. Since about 47 per cent. of drivers involved in accidents appear to be in the habit of drinking, it would appear that the liability to accident of the drinking driver is considerably greater than that of the non-drinking driver. Since the percentage of alcohol in the blood of each driver was known, it would be of interest to find out at what concentration the chances of accident in the non-drinking group approximated that in the drinking group. It was found that the accident-liability increased as the amount of alcohol in the blood increased, as would naturally be expected, but that when the concentration of alcohol was 0·05 per cent., or lower, there was no increased tendency to accident. In other words, there was the same chance of accident in drinking and non-drinking drivers when the former had 0·05 per cent. or less alcohol in the blood. This indicates that at that concentration, which represents about one large whisky, alcohol is not a significant cause of accidents. Holcomb calculated that a driver with 0·15 per cent. alcohol in his blood had fifty-five times as great a chance of meeting with an accident as one free of alcohol, and if his data are correct, they illustrate the importance of the problem and the necessity of dealing with it.

As a general conclusion on this aspect of the subject, it may be accepted :—

(1) That the part played by the so-called drunken motorist in road accidents has been greatly exaggerated.

(2) That the ingestion of alcohol exercises a deleterious influence on driving and is a potential danger in quantities which do not produce sufficient effect to be recognisable by clinical examination.

(3) That the clinical examination for alcohol in the blood can give a reasonably accurate idea of the total amount ingested and, although in itself it cannot prove that a person is incapable of having control of a motor, it can indicate that the skill of the person driving is reduced.

(4) That the point at which alcohol induces dangerous driving cannot be laid down for any one individual, but there is sufficient evidence to indicate that at or about 0·125 to 0·15 per cent. in the blood, there is a definite danger. This does not mean that below that figure the driver is not affected ; he may be clinically intoxicated with less than 0·1 per cent., but the position becomes critical at that concentration.

(5) That at a concentration of 0·15 per cent. the *majority* of people are intoxicated, but some are not and therefore, although no rigid interpretation of intoxication is possible from the test, it can quite properly be used as a corroborative test.

(6) That if a definition used in a number of States in the U.S.A. were adopted in this country, namely :—

" If the ability of a driver has been lessened in any degree by the use of intoxicating liquors, then the driver is assumed to be under the influence of intoxicating liquor." then the problem of dealing suitably with the drinking driver would be greatly simplified.

Alcoholic Coma. The insensibility produced by alcohol may not come on until after a certain period, when it may develop with great rapidity. Christison met with an instance in which a person fell suddenly into a deep stupor, some time after he had swallowed sixteen ounces of whisky, without any of the usual premonitory symptoms. In another

instance, a person may apparently recover from the first effect, and then suddenly become insensible and die convulsed. In the paralytic stage the respiration is slow and stertorous, the pulse almost imperceptible, the skin cold and cyanatic. The pupils may be contracted, dilating on stimulating the skin, or in some cases dilated and fixed. The reflexes may be absent. The more concentrated the alcohol, the more rapidly are the symptoms induced, and the more severe is their character. Dilute alcohol generally produces a stage of excitement before stupor, while concentrated alcohol may produce profound coma in a few minutes.

This **coma requires to be differentiated** from apoplexy, concussion, and other forms of coma due to uræmia, diabetes, etc., and in connection with the latter it is to be remembered that an overdose of insulin may cause coma from hypoglycæmia. In any case of genuine insensibility or coma, the condition of the patient is serious, and he *must not* be left in a police cell, but must be placed under careful supervision and treatment, **either at home or in a hospital.**

(1) The history, if obtainable, may decide the matter offhand.

(2) **Local injury to the head should be excluded.**

(3) Note the pupils, dilated in alcohol poisoning and in many other poisons. According to MacEwen, if the patient has lain unmolested for half an hour the pupils will be found contracted: if any external stimulation is applied, such as moving him, pulling his hair or slapping him, the pupils will be seen to dilate gradually. On allowing him to lie still the pupils again contract. A contracted pupil may indicate opium poisoning or hæmorrhage into the pons: unequal pupils, hæmorrhage into or injury of the brain; and if the pupils are inactive the case is **serious, whatever be its precise nature.**

(4) Temperature: raised—hæmorrhage into pons: lowered—signifies *danger*, **but does not differentiate the source of it.**

(5) Localised paralysis, as opposed to general helplessness, suggests an asymmetrical lesion, probably hæmorrhage or softening.

(6) The urine should be examined for alcohol, sugar, acetone, albumen, **casts, blood, etc.**

(7) The general appearance of the skin: flushed and sweating (probably alcohol), cold and blue (collapse from other poison), or cold **and sweating (opium).**

(8) The breath may be smelt. If no odour of alcohol, it is practically certain that the case is not "drunk": if an odour, this is not of much value without some history of how, or why, the alcohol was taken, and it excludes neither apoplexy nor any other serious condition. A smell of opium, carbolic, or any definite drug other than alcohol, is strong **presumption in favour of poison.**

The other, infinitely more difficult aspect of this question is that in which a **condition of excitement or active stupidity,** really due to gross organic disease, and shortly to be followed by a fatal coma, is mistaken for the pre-comatose stage of drunkenness. It is mistakes made in this direction that have so frequently led to severe strictures being passed by the public press on private practitioners, on police surgeons, and on **hospital resident medical officers.**

To clear up the matter precisely, the same observations should be made as in the cases of coma, but there are difficulties in the way in

that (1) the person concerned may resent the examination by violent struggles or otherwise, and (2) he may have been observed only at a distance performing stupid actions which are attributed to drunkenness, but are in reality due to serious disease. In the former case he must be kept under careful professional attention till an examination can be made; in the latter case a medical witness must be firm, but cautious, in the deductions he draws from his *post-mortem* examination (*vide*, for such a case, Vol. I, under "Head Injuries").

Treatment. In the ordinary forms of alcoholic intoxication, nothing is required but to let the patient sleep it off under observation. If symptoms are severe the stomach should be thoroughly washed out with warm water, and bicarbonate of soda and glucose may be administered intravenously if the condition warrants it. It is the activity of this treatment compared with the measures required for apoplexy, for poisoning, for meningeal hæmorrhage, or for other acute cerebral affections that renders the diagnosis so important.

Palthe[1] found that inhalation of oxygen had a powerful antagonistic effect on the toxic effects of alcohol. In experiments on rabbits it was possible to counteract the effect of a lethal dose of alcohol by such inhalation. In human beings the symptoms were much reduced, and a very favourable effect was produced on the motor and mental symptoms in two cases of delirium tremens in which it was tried.

Methyl Alcohol (CH_3OH). "Wood Spirit" is prepared in enormous quantities by the destructive distillation of wood. The crude "pyroligneous acid" consists of acetic acid, wood alcohol, aldehydes, ethers, ketones and other products. The acetic acid is separated out as a salt, and the wood alcohol is collected. A great deal of this comes to Europe from America under the name "American Crude Wood Alcohol," its composition being: acetone and other ketones, 16 per cent.; aldehydes, amines and oils, 6 per cent.; methyl alcohol, 70 per cent.; water, 8 per cent. The alcohol is separated by fractional distillation and yields "denaturing wood alcohol," used everywhere for denaturing alcohol. From this other grades of methyl alcohol are prepared, varying in purity. In 1853 the British Government sanctioned the use of "methylated spirits" duty-free, this consisting of a mixture of 9 parts of spirits of wine and 1 part of methyl alcohol (wood naphtha). In 1891, owing to the possibility of "methylated spirits" being sufficiently purified to make it potable, and the growing practice of drinking even unpurified methylated spirits especially by the lower classes, it was decided to restrict the use of methylated spirit to manufacturers only and to prescribe the addition of a further denaturant for general purposes. This denaturant consists of mineral naphtha (petroleum), and the mixture known as "mineralised methylated spirits" is sold to the general public. It was found, however, that ordinary methylated spirits was not universally applicable to manufacturing processes requiring the use of alcohol, and accordingly in 1902 the Commissioners of Inland Revenue authorised the use of duty-free alcohol denatured with substances other than wood naphtha, and in 1904–5 the amount of wood naphtha to be used for denaturing "ordinary" methylated spirit for industrial purposes was reduced from 10 to 5 per cent. of the mixture. According to the Methylated Spirit Regulations, 1930, the following varieties of methylated spirit are recognised.

Mineralised Methylated Spirits consisting of 90 per cent. by volume of ethyl alcohol, 9½ per cent. of wood naphtha and ½ per cent. of crude pyridine. The mixture to be coloured with an aniline dye (Methyl Violet).

Industrial Methylated Spirits consisting of 95 per cent. by volume of alcohol and 5 per cent. of wood naphtha.

Industrial Methylated Spirits (pyridinised) consisting of industrial methylated spirits with the addition of ½ per cent. of crude pyridine.

[1] *Deutsche Zeitschrift für Nervenheilkunde*, Leipzig, July 1926.

Power Methylated Spirits containing 1½ per cent. wood naphtha, ½ per cent. crude pyridine, 5 per cent. petrol and red colouring matter.

Retailers of methylated spirits may sell only mineralised methylated spirits, but under Regulation 50 a wholesale or retail chemist may obtain authority to sell industrial spirits in quantities of not more than ½ gallon at a time to a medical practitioner, dentist, veterinary surgeon, hospital or nursing home on receipt of a signed written order.

In many parts of the country methylated spirit is used for drinking purposes in spite of the addition of the nauseous substances prescribed by the Regulations. The spirits are mixed with aromatic mineral waters and with the cheaper varieties of red wine. The latter beverage, known as Red Biddy, makes a potent and not particularly unplalatable drink.

Pure methyl alcohol, made synthetically or by refining wood alcohol, is a colourless mobile fluid with an odour similar to ethyl alcohol, and has a burning taste. It is used for denaturing ethyl alcohol, as a solvent for fats and varnishes, for cinematograph films, for the production of formalin and other chemicals, and in many other trade processes. It has been used considerably in the adulteration of alcoholic beverages, especially in the United States of America. The drinking of pure methyl alcohol is not common, though cases occasionally occur among workers in chemical factories. Although a large number of known cases have been investigated in this country, the typical effects of methyl alcohol on the optic nerve have been observed in comparatively few. In the United States of America and in Germany a very large number of cases of blindness have been reported, and Leschke (*Münch. Med. Woch.*, April 29th, 1932) states that no less than 400 deaths occurred from its use in America in the winter of 1931.

Methyl alcohol is readily absorbed into the body, but though its action resembles that of ethyl alcohol to a great extent, it is eliminated much more slowly, and therefore with repeated small doses tends to accumulate in the blood. Windhausen[1] states that it does not disappear from the blood until about three or four days, and gives the fatal dose for man as under 120–240 grams. Symptoms may be delayed for several hours.

The symptoms of poisoning by the drug consist of headache and nausea, vomiting and pain or severe cramps in the abdomen. There is marked muscular weakness and depressed cardiac action. There may be dyspnœa and cyanosis. The effect on the central nervous system is more intense and persistent than with ethyl alcohol. There may be delirium and coma, which may last for two or three days. There is a toxic effect on highly specialised nerve elements, and partial or complete blindness is a common sequel.

It has been suggested that many of the toxic features of wood alcohol are due to the impurities which it contains, and that synthetic methyl alcohol, which is pure, is free from these effects. This is untrue, for Hunt[2] and other investigators have shown that pure methyl alcohol is just as toxic as wood alcohol. It has been suggested that the formation of formaldehyde and formic acid during the oxidation of methyl alcohol is the true cause of the toxic phenomenon. Roe[3] in the investigation of 16 cases came to the conclusion that the symptoms were due to acidosis and varied with the fall of the alkali reserve. He found that ethyl alcohol asserted a favourable effect. In treating a case, the stomach must be thoroughly washed out, and intensive treatment with alkalies adopted to combat the acidosis.

Fusel Oil. The mixture of alcohols constituting this substance is produced in comparatively small quantity in the alcoholic fermentation of most bodies containing sugar, the largest proportion being contained in alcohol prepared from potatoes. Fusel oil consists mainly of two alcohols, one of which, *iso*-butyl-carbinol or inactive amyl alcohol $(CH_3)_2CH.CH_2CH_2.OH$), is optically inactive, while the other, active amyl alcohol $(C_2H_5(CH_3)CH.CH_2.OH)$, is strongly lævorotatory. Formic, acetic, propionic, butyric, valeric and other acids, ethyl acetate and other esters, are also present.

The injurious effect of raw or recently manufactured spirits is attributed in part at any rate to the presence of fusel oil produced during the fermentation and not completely separated, but it is not known whether this is really so or not.

[1] *Chem. Zeitung*, 1926, 50, 558.
[2] *Amer. Jour. Pharm.*, 1925, 97, 495.
[3] Roe, D., *Acta med. Scand.*, 1943, 113, 558.

In England it goes by the name of "faints," and can be obtained gratis from some distilleries. Fusel oil is an oily liquid with a burning acrid taste and an odour said to resemble jargonelle pear, and is popularly used as an external application for rheumatism. It has intoxicating and poisonous properties superior to those of ordinary spirits, and is said to be known in some of the northern countries of Europe to the consumers of corn brandy, who frequently ask to be served with a "glass of good fusel."[1]

Cases of alcohol poisoning are usually accidental; these bodies can hardly be used for homicidal purposes, and assuredly not for suicide. Alcohol may act as a poison by its *vapour*. If the concentrated vapour be respired, it will produce the usual effects of intoxication. There is a case on record in which a child two years of age was thrown into a stupor by the alcoholic vapour of eau de Cologne.

Post-mortem Appearances. The inspector must be careful to smell the contents of the stomach, though it is possible that the smell may have disappeared. A sample of the stomach contents and a sample of blood should be taken for analysis in every case.

Alcohol undoubtedly acts as an irritant of mild degree to the stomach. This organ may be congested or inflamed or there may be little change from the normal. When death has taken place rapidly, there may be the peculiar odour of some form of spirits in the contents; but this will not be perceived if the quantity taken was small, or if many hours have elapsed before the inspection is made. The brain and its membranes are sometimes found congested.

A girl was found at four o'clock in the morning lying insensible on the floor. She had had access to some brandy, which she had swallowed from a quartern measure found near her quite empty. She had spoken to her mother only ten minutes before, so that the symptoms must have appeared very rapidly. She was seen four hours afterwards. She was then quite insensible, in a state of profound coma, the skin cold and covered with a clammy perspiration. There had been slight vomiting. The child died within twelve hours, without recovering consciousness, from the time at which she was first found.

On inspection, there was injection of the brain and its membranes; the heart and lungs were quite healthy. The mucous membrane of the stomach presented patches of intense redness, and in some places it was thickened and softened; portions of it were detached and hanging loosely in the stomach; and there were patches of black extravasated blood about it. It contained a greenish-coloured liquid, but there was no smell of brandy in it, neither was this perceptible in the breath of the child, four hours after the alcoholic liquid had been taken. At first it was suspected that arsenic had been administered; but the symptoms were not those of arsenical poisoning, and neither arsenic nor any other metallic irritant was present in the contents of the stomach. Traces of alcohol were detected by the process described below.

Analysis. When a large dose has been taken and the case has proved rapidly fatal, the contents of the stomach may have the odour of alcohol, or of the alcoholic liquor taken. The odour is not always perceptible, or it may be concealed by other odours. In a case of poisoning by gin, the liquid withdrawn from the stomach after seven hours had no odour. In the case of the child already related, the smell of brandy had entirely

[1] *Lancet*, 1901, 2, p. 606.

disappeared in twelve hours. The contents of the stomach or the suspected liquid should be distilled in a water-bath, with a proper condensing apparatus attached. The watery liquid obtained should be mixed with anhydrous sulphate of copper or of sodium in sufficient quantity, and submitted to a second distillation in a smaller flask, by a water-bath. The liquid resulting from the second distillation should be shaken with rather more dry carbonate of potassium than it will dissolve, in a small tube provided with a stopper, and allowed to stand. A layer of alcohol, if present, will, after a time, float on the surface, and may be drawn off by a pipette and examined. *Tests.* 1. Alcohol has a hot pungent taste, a peculiar odour, and is very volatile. 2. Absorbed in asbestos, it burns with a pale blue flame, which deposits no carbon on white porcelain; and when burnt in the mouth of an inverted test-tube, containing a few drops of baryta, or lime-water, it produces a well-marked white deposit of carbonate of barium or calcium. Carbonic acid and water are the sole products of its combustion. 3. The liquid becomes green when boiled with a few drops of a solution of bichromate of potassium mixed with sulphuric acid. 4. The alcoholic liquid is made alkaline with a solution of potash or soda, warmed, and a solution of iodine in iodide of potassium is added until the liquid is brownish yellow; potash is again added till the liquid again just becomes colourless. Sooner or later iodoform separates as a yellow scaly precipitate, which, when examined under the microscope, is seen to consist of six-sided stars and rosettes. 5. The alcoholic liquid is warmed with a little glacial acetic acid and a few drops of concentrated sulphuric acid. Ethyl acetate is formed and recognised by its "fruity" odour.

The following method will allow of the detection of a quantity of alcohol too small for separation by the process above mentioned. Make a mixture of strong sulphuric acid and a saturated solution of bichromate of potassium: moisten with this mixture a few fibres of asbestos, and enclose them in a glass tube connected with the retort or flask in which distillation is carried on. For this purpose a flask or tube similar to those used for detection of chloroform vapour will be found serviceable. The smallest portion of alcohol-vapour passing over the asbestos, immediately renders it green, by converting the chromic acid into chromic sulphate. This may serve as a trial test or for evidence, according to circumstances. The tube may be removed, and the condensed vapour collected for the application of the other tests. Ether and methyl alcohol produce a similar result, as do other volatile oxidisable organic compounds (aldehydes, ketores, etc.).

Colorimetric Method of estimating Traces of Alcohol in Dilute Solution. The method is based upon the following facts: If we boil for one minute a mixture of 1 c.c. of 2 per cent. solution of potassium bichromate, 5 c.c. of 0·1 per cent. solution of alcohol, and 5 c.c. of concentrated sulphuric acid, allowing the mixture to cool, the liquid assumes a pure green colour (chromic sulphate).

A series of tints varying from green to yellow can be obtained from the following quantities of the 0·1 per cent. alcohol solution: 5 c.c., 4·5 c.c., 4 c.c., 3·5 c.c., 3 c.c., 2·5 c.c., 2 c.c., 1·5 c.c., 1 c.c., and 0·5 c.c., completing each volume to 5 c.c. with distilled water, and then boiling with the potassium bichromate and sulphuric acid as stated above.

A tube with 5 c.c. water in place of the 5 c.c. of the 0·1 per cent. alcohol has to be taken in the series as a control.

These standards can be kept for a long time if the tubes are covered to prevent dust and evaporation. The difference in colour in the series is quite distinct, and with practice values of the intermediate stages can be estimated with sufficient accuracy.

From the solution of alcohol under examination, 5 c.c. are taken, and 1 c.c. of the bichromate and 5 c.c. of concentrated sulphuric acid are added; the mixture is boiled for one minute, and cooled. The colour is then compared with the standards. Intermediate colours between green and yellow can at once be read. If the colour is pure green the mixture contains *at least* 0·1 per cent. alcohol. A smaller quantity is then taken, diluted with water to 5 c.c., and similarly examined, until a comparable tint with one of the control tubes is obtained.

When the process is applied to blood or urine, a preliminary distillation is necessary. A measured volume of blood mixed with a little picric acid, or of urine made alkaline with sodium hydroxide, is submitted to steam distillation in a large flask. An efficient condense is required and the receiver, a graduated flask or cylinder, should be cooled in ice. A volume of distillate exactly equal to the volume of fluid taken for the analysis is collected, and 5 c.c. of this are used for the development of the colour as already described.

Cannan and Sultzer's Method for the determination of alcohol in the blood[1] :—

A known volume of blood is delivered directly on two or three times its weight of anhydrous sodium sulphate, which is distributed over the bottom of a distilling flask.

This is placed on the water-bath at 40° to 50° C., and evacuated through a tube containing a known volume of standard potassium dichromate, and an equal volume of strong H_2SO_4. The distillation is allowed to proceed for fifteen to twenty-five minutes with the pump running. The vacuum is then broken by opening the capillary inlet of the distilling flask and the absorption tube disconnected. The contents are washed into a flask with sufficient water to dilute the H_2SO_4 to less than 5 per cent., excess of 10 per cent. potassium iodide solution is added, and the liberated iodine is titrated with standard sodium thiosulphate and starch. This titration, subtracted from the thiosulphate titre of the volume of potassium dichromate used, gives the amount of the latter required to oxidise the alcohol, and from the factor, 1 c.c. $N/10\ K_2Cr_2O_7 = 1{\cdot}15$ mgrm. alcohol, the amount of alcohol can be obtained. It must be borne in mind that in these procedures all volatile reducing substances are included in the estimated "alcohol." The editor has found that perfectly normal blood may appear to contain as much as 50 mgrm. of alcohol per 100 c.c. The results obtained by this method are probably always a little too high. A convenient apparatus, enabling the estimation to be made on 2 c.c. of blood has been described by Southgate (Carter and Southgate, *Trans. Medico-Legal Soc.*, Vol. 20, p. 31).

[1] "Heart," 1924, 148.

Widmark's Method.[1] Blood is collected from a finger-prick into a capillary tube. Note that no alcohol, ether, or other volatile reducing agent may be used in cleansing the skin or in any syringe or needle used for obtaining the blood—precautions which apply whatever method of estimation is being used. The tube is weighed, and the blood is transferred to the cup (A) of the apparatus. The tube is reweighed, and the weight of blood taken for analysis is thereby found; it should be 100 to 200 mg. The flask contains 1 c.c. of the bichromate-sulphuric acid reagent (0·1 g. or 0·25 g. pure $K_2Cr_2O_7$ dissolved in 1 c.c. of water and made up to 100 c.c. with concentrated sulphuric acid; the more dilute reagent is used when the expected alcohol concentration is below 0·2 per cent.). The ground-glass stopper carrying the cup A is fitted to the flask and held in place by a rubber cap. The flask is then heated in a water-bath at 50–60° C. for two hours. The stopper is removed with great care (particles of dried blood must not be allowed to fall from the cup), the reaction mixture is diluted to about 25 c.c. with distilled water, 0·5 c.c. of 5 per cent. KI solution is added, and the mixture is titrated with 0·01 N. sodium thiosulphate solution. The volume of thiosulphate (in c.c.) is subtracted from the volume required for the reagents alone. The difference, multiplied by 0·113 (an empirical factor since the alcohol is not oxidised according to the theoretical equation), gives the weight of alcohol, in mg., present in the blood.

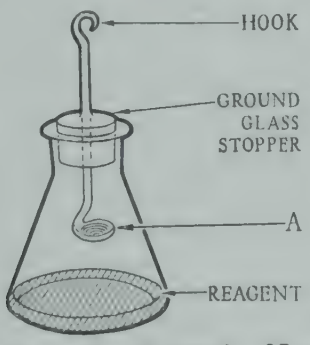

Fig. 7.—Widmark's Flask.

Method of Friedemann and Klass.[2] None of the methods just described, is specific for ethyl alcohol; any volatile reducing substance will interfere. In blood, acetone and acetoacetic acid are the most likely interfering substances and in severe ketosis may, as Widmark pointed out, account for as much as 0·03 per cent. (or, rarely, much more) of apparent alcohol. In urine, unless the fluid is made alkaline volatile organic acids may also interfere.

These difficulties are at least partly overcome in the method of Friedemann and Klaas, in which a preliminary treatment with mercuric oxide remove interfering aldehydes, etc., and alkaline permanganate is used instead of acid bichromate as the oxidising agent.

For blood, saliva, tissues (frozen in liquid air and crushed), a sample of about 1 g. is introduced into a 300 c.c. Kjeldahl flask, mixed with about 50 c.c. distilled water, 5 c.c. of sod. tungstate (10 per cent.) solution, 5 c.c. of mercuric sulphate (10 per cent. in 2 per cent. sulphuric acid), a little powdered talc and brought slowly to boiling point. In about twenty minutes, 30 to 35 c.c. are distilled, the vapour passing through a tinned metal condenser to a well-cooled recever.

[1] *Biochem. Z.*, 1922, *131*, 471.
[2] *J. Biol. Chem.*, 1936, *115*, 47.

For urine, this procedure is carried out, making the initial volume 100 c.c. and collecting 60 c.c. of distillate in a Kjeldahl flask containing 5 c.c. of the mercuric sulphate solution. To this receiver, enough 20 per cent. suspension of calcium hydroxide is added to give a deep orange colour, powdered talc is added to prevent foaming, and 30–35 c.c. are distilled off into a well-cooled receiver.

The whole, or an aliquot part (made to 35 c.c. with distilled water) of the distillate is mixed with 10 c.c. of 5 per cent. sodium hydroxide and, with constant rotation, 25·0 c.c. of standard potassium permanganate solution (0·02 N, 0·01 N or 0·005 N according to the amount of alcohol expected). The mixture is heated in a boiling water-bath for twenty minutes and then cooled in running water, 10 c.c. of 10 N sulphuric acid are added, and the mixture is titrated with standard sodium thiosulphate. Blank determinations must be done on the reagents. The titration should be made with thiosulphate of the same normality as the permanganate used. The quantity of alcohol should be such that the volume of permanganate used to oxidise it is not greater than 5 c.c. Oxidation is not complete and the following empirical factors are used :

1·00 c.c. of 0·22 N $KMnO_4$	=	0·0855mg. ethyl alcohol
1·00 ,, ,, 0·01 N ,,	=	0·0420 ,, ,, ,,
1·00 ,, ,, 0·005 N ,,	=	0·0215 ,, ,, ,,

From lapse of time, the effects of treatment, or absorption and elimination, there may be no trace of alcohol in the stomach or intestines ; nevertheless the person may have died from the effects. In one case, fatal in eight hours, no alcohol was found in the stomach. One cause of failure may sometimes be traced to the distillation being restricted to a portion of the contents. It is advisable to distil the *whole*, as, if necessary, the distillate or the residue can be examined for other poisons. Distillation of a sample of blood should be carried out in every case of alleged poisoning by alcohol.

Fusel Oil is a volatile liquid of a pale yellow colour, lighter than water and only sparingly soluble in it. It is dissolved by alcohol and ether in all proportions, but not readily by chloroform. Water separates it from its ethereal solution. It has a hot burning taste and an offensive spirituous odour, which is very persistent and peculiar : by this it may be distinguished from other alcoholic liquids. It is inflammable, and burns with a pale bluish flame. Like alcohol, ether, and wood-spirit, it decomposes chromic acid, producing green chromic oxide (or chromium sulphate if sulphuric acid is present). In organic mixtures ether might be used for its separation.

By distilling one part of fusel oil with two parts of acetate of potassium and one part of sulphuric acid, an ethereal liquid, amyl acetate, is produced, which is used in confectionery under the name of *Essence of Jargonelle Pear*. Since the odour of this ester is characteristic, its preparation may be used as a test for fusel oil. A child on two occasions became partially comatose and had livid lips and a feeble pulse, after eating some confectionery which it was calculated contained about one drop of this essence. Hence its use is not without danger.[1]

[1] *Pharm. Jour.*, November 1851, p. 214.

Methylated Spirit. It is a very inflammable liquid, burning with a pale blue flame. It is light and volatile, readily in part separable from other liquids by distillation below 200° F. Its odour is peculiar. It mixes with water and alcohol in all proportions.

Methyl alcohol reduces acidified potassium dichromate to green chromic sulphate just as ethyl alcohol does. Whereas ethyl alcohol, however, produces acetaldehyde, methyl alcohol produces formaldehyde. If, therefore, the suspected material be distilled and the distillate be again distilled with potassium dichromate and sulphuric acid, using a well-cooled condenser and receiving vessel, a solution of formaldehyde is obtained. To this solution a few drops of a 5 per cent. solution of phenyl hydrazine are added, followed by one drop of 0·5 per cent. sodium nitroprusside solution and a few drops of 10 per cent. sodium hydroxide solution. In presence of formaldehyde, a blue colour appears, changes to green, and finally a yellowish-red. (Acetaldehyde gives a red colour.) Other tests for formaldehyde may also be applied.

Cases. At an inquest held in May 1904, the evidence showed that a Montgomeryshire farmer gave his son, aged four, whisky to drink, with the result that the child died in convulsions. The jury returned a verdict of death from alcoholic poisoning, and the father, who admitted that his children were frequently given beer and spirits at home, was censured.

A woman, *æt.* 41, drank straight off one and a half pints of whisky twenty-two overproof; she died within five and a quarter hours.[1]

Two pints of whisky caused death within eight hours in an adult.[2]

A child, *æt.* 3, died comatose after a small tea-cup of whisky,[3] and a boy of six after three ounces of whisky was seized with spasms and died with a temperature of 105° F.[4]

On July 23rd, 1926, a London physician was arrested and charged with being drunk while driving a motor car.

It was stated in evidence that while driving at midnight the car mounted the footpath, ran along it for some distance, then turned across the road, ran into a lamp-post, rebounded across the road, and came to rest against a garden wall.

The constable who made the arrest gave evidence that the accused was drunk, and this was confirmed by the police-surgeon. The accused gave evidence that he was not suffering from the effects of alcohol, but from the effects of an overdose of insulin. He was supported by four doctors, who all stated that the effects of insulin might be mistaken for drunkenness.

He was convicted, fined, and his licence withdrawn for twelve months. Twelve days afterwards he died from diabetes.

It should be understood that unless insulin is given with care, not only as regards the dose, but also as regards the next meal, there may be loss of nervous and muscular control due to the consequent hypoglycæmia, which may readily be mistaken for the effects of alcohol.

On poisoning by fusel oil, the following is taken from the *Lancet*, 1901, 2, p. 606:—

"Dr. Thomas B. Futcher, of Johns Hopkins University, records in the *New York Medical Journal* of August 3rd two cases from the clinic of Professor Osler of the somewhat rare condition of poisoning by 'fusel oil,' an ingredient of crude spirits. The cases recorded in medical literature are very few. Dr. W. M. Ord reported a case at St. Thomas's Hospital in 1889,[5] and Dr. J. Swain[6] recorded a fatal case at the Bristol Royal Infirmary in 1891. The two cases recorded by

[1] *B.M.J.*, 895, 1, p. 72.
[2] *Lancet*, 1897, 1, p. 1158.
[3] *Ibid.*, 1899, 2, p. 63.
[4] *Ibid.*
[5] *Lancet*, 1889, 2, p. 1225.
[6] *B.M.J.*, 1891, 1, p. 903.

Dr. Futcher are as follows : —Case 1 was that of a woman, aged twenty-eight years, who was admitted to the Johns Hopkins Hospital at about midnight, complaining of general weakness. It was found that in company with a male (Case 2, *infra*) she had been drinking out of a six-ounce bottle of fusel oil during a debauch and was lying unconscious on the floor. At 9 a.m. next morning she had a headache, coated tongue, and a bad taste in the mouth and suffered from general weakness. Her forehead was red from rubbing it, as she stated, with fusel oil a day earlier. There was a slight bronchitis. The pupils were normal in reaction, and an estimation of the blood showed 4,500,000 red corpuscles and 7,000 leucocytes per cubic millimetre. The urine was of a pale yellow colour and slightly hazy, with a white precipitate, and exhaled ' a peculiar odour resembling violets.' There was a faint trace of albumen, and on testing with Fehling's solution sugar was found to be present. Tests with phenylhydrazine and with yeast confirmed the presence of sugar, but the polariscope showed that it was not glucose (dextrose), as the solution was laevo-rotatory. Next day the urine was free from sugar, but a trace of albumen persisted for a day or two longer. She was discharged as recovered after four days. Case 2 was that of a man, aged twenty-eight years, who was admitted into the hospital at the same time and under the same conditions as the above patient. His temperature was 100° F., the pulse was 120, and the respirations were forty-eight per minute. The right arm showed impairment of sensation and the right leg showed loss of power and an excessive knee-jerk. He gradually improved in hospital, though the muscular and sensory impairment persisted for four or five days. He was discharged on the sixth day. His urine had a dark brown ' smoky ' appearance, with a ' peculiar aromatic fruity odour.' It gave the same chemical reaction for sugar as did the urine in the previous case, but was inactive to polarised light. Methæmoglobin was also present in the urine with a few disintegrated red corpuscles. The sugar disappeared from the urine during the second day. The presence of sugar in both these cases was a peculiar and very rare feature."

Poisoning by Methyl Alcohol (*Wood Spirit*), *Epidemic in Berlin*. On the morning of December 27th, 1911, a number of people in the Municipal Shelter for the Homeless were suddenly seized with illness and fifteen died. Twenty more were so ill that they were removed to the Friedrichshain Hospital. Other cases occurred, in the end amounting to 129, of which ninety-four were very severe and fifty-seven died. The Shelter was a refuge for people having no abode and persons were admitted without formality for the night. The symptoms of those affected were very striking, and included profound cyanosis of hands, feet and face. There was manifestly a high degree of air hunger and excessive restlessness, the patients throwing themselves about in their beds and boring their heads into their pillows. Convulsions and tremors were the rule, and most complained of great pain in the joints and belly. The temperature was subnormal. Ocular symptoms were present in almost all the affected, and included flashes of light, dimness of vision amounting to complete amblyopia and great dilatation of the pupils. In the fatal cases the symptoms rapidly increased, the extremities were ice-cold and perspiring, and with clouded mind deepening into coma, complete paralysis of the respiratory centre completed the picture. The autopsies showed especially hæmorrhages in the meninges and acute changes in ganglion cells of brain and cord.

At first it was supposed to be an epidemic of food poisoning of a botulismus type, specially as there was a street traffic of cheap comestibles in the neighbourhood of the Shelter, the inmates buying a goodly part of their food outside the institution It was, however, ultimately proved that the real cause of the poisoning was cognac adultered with methyl alcohol. When the public-houses round the Shelter were closed the epidemic ceased. The wholesale dealer from whom the adulterated cognac was obtained was traced, and in his cellars the police found great quantities of methyl alcohol, a portion of which he had sold as genuine ethyl alcohol. The accounts of the above epidemic vary somewhat in the different publications. In some it is stated that the number attacked was 171, of whom seventy-eight died. The account abridged above is that by Stadelmann, Chief Physician to the Friedrichshain Hospital.

The toxic effect of wood spirit and methyl alcohol has been recognised for years, especially in America, Russia and Hungary. In Buller and Wood's monograph a full account of the symptoms is given, special attention being drawn to the ocular phenomena. There were 153 cases of blindness, of whom 122 died.

The toxic effects appear to be due to pre methyl alcohol and not specifically to the impurities in the cruder unrefined varieties known especially under the name of " wood spirit " although of course such impurities may have an added effect.

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Poisoning by Paraldehyde $(CH_3CHO)_3$

Source and Method of Occurrence. This drug, of a very nauseous taste and strong odour, is official, and its dose as a hypnotic is given as thirty to one hundred and twenty minims. On its introduction it was thought

to be an absolutely safe hypnotic ; but no such thing does nor can in the nature of things exist. It was also thought that its nauseous taste would prevent anyone acquiring a habit of taking it ; but this hope has likewise proved fallacious.[1]

It is used by the mouth and by the rectum. It has been used for basal anæsthesia intravenously and per rectum.[2, 3]

Toxicity and Fatal Dose. It must be admitted that its toxic powers are but feeble, and that as a matter of fact it is a fairly safe hypnotic in so far that its effects on the heart are stimulant rather than depressant : thirteen drachms[4] and three and a half ounces[5] have been given without fatal effect, but, on the other hand, two ounces have proved fatal[6] and deaths have been reported from as little as 25, 31, and 52 c.c., of the drug.[7]

A recent case of death from taking two and a half to three ounces is recorded,[8] the victim in this instance being a man who was in the habit of taking teaspoonful doses of the drug. He went to bed at 11 p.m., and was found dead at 8 a.m.

Symptoms. Its action is fundamentally similar to that of alcohol but has a pronounced hypnotic effect causing profound sleep which with toxic doses deepens into coma. Of other symptoms vomiting, nausea, and giddiness are the most prominent. Its continued use leads to symptoms similar to chronic alcoholism including attacks of delirium tremens and ultimate mental and moral deterioration.

Cases. In 1891, a supposed case of suicide by paraldehyde was communicated to Sir Thomas Stevenson by Bond. A lunatic, to whom this substance was being administered, managed to retain his nightly dose in the mouth and secrete it till he had obtained such a quantity of paraldehyde, as to cause fatal effects when taken in one dose.

Mackenzie was called to a patient who had taken three and a half ounces of paraldehyde. The breath smelt strongly of the substance ; the face was slightly flushed, the pupils moderately contracted and quite insensible to light ; the breathing and pulse were rapid, the skin warm. The woman was quite unconscious, and her body absolutely limp, like an anæsthetised person. She recovered.

The following is from the *Lancet*, 1902, 2, p. 673 :—

A woman was ordered one drachm each of paraldehyde and syrup of orange-peel and water to one ounce ; but by misadventure she was given an ounce of pure paraldehyde at 12 midnight, and the error was neither discovered nor even suspected until some seven hours afterwards. In a quarter of an hour the breathing became somewhat irregular and the patient's mouth and larynx full of a thick ropy mucus ; the latter was all swabbed out and the breathing then greatly improved. As the patient was also suffering from bad stomatitis, it was now thought that some of the draught (as ordered) had entered the lungs during the struggling when it was administered. At 1.15 a.m. the breathing was still rapid and irregular, thirty-two respirations to the minute, but the pulse was fairly strong (114) ; the patient, however, half choked with mucus, was becoming somewhat cyanosed, and the tongue was very red and swollen. The mouth was again swabbed out with relief. The patient was now semi-conscious, fairly sensitive to pain, but made no voluntary movement. However, she was easily roused by shouting, pinching, etc. The pupils were variable in size, but reacted both to light and to accommodation. At

[1] Carver, A. E., *Lancet*, 1 : 408 (1934).
[2] Whigham, J. R., *B.M.J.*, 2 : 320 (1935).
[3] Nitzescu, *Presse Medical*, 1 : 331 (1934).
[4] *Lancet*, 1900, 2, p. 875.
[5] *B.M.J.*, 1891, 2, p. 1254.
[6] Westcott, " Extra Pharm.," 1904, p. 65.
[7] Kotz, *et al.*, *J. A.M. A.*, 110, 2145 (1938).
[8] *B.M.J.*, August 8th, 1925.

2.15 a.m. she was given a hypodermic injection of three minims of liquor strychninæ with two minims of tincture of digitalis, which greatly improved both the respiration and cyanosis, but half an hour later the breathing again became rapid and irregular, with long inspiration and short expiration. The lips were pale and the mouth was dry and free from mucus. The pulse was regular (124), but the patient was quite unconscious. The pupils were contracted and the corneal reflexes were completely lost. At 3.30 a.m. the breathing was still shallow, more ropy mucus appeared in the mouth, the pulse was still 124, but the patient was more conscious and slightly sensitive to pain ; at this time there was an involuntary slight action of the bowels. After this she became somewhat restless, tossing about and moaning occasionally ; the pupils were dilated but not reacting to light. The pulse was good, and the respirations were much quieter, continuing in this condition until 7.30 a.m., when she again became cyanosed, the pulse feeble and rapid, and the skin hot and dry, with very shallow respirations. At this period of the case the unfortunate mistake in the composition of the draught was discovered, but it was then deemed too late to give emetics or to wash out the stomach, so amyl nitrite was at once inhaled and five minims of liquor strychninæ were given hypodermically, followed by a hot coffee enema ; these remedies greatly improved both the pulse and respiration, but the patient still remained somewhat cyanosed, with rapid and shallow breathing. She was then given diffusible stimulants in the shape of ammonia and ether, with brandy and hot water by the mouth, but as these were swallowed with difficulty brandy (twenty minims) was subcutaneously administered. For some hours she continued in a very drowsy state, the temperature reaching 100° F., with a pulse rate of 116 and a respiration rate of twenty-eight. Still being unable to swallow anything she was given a nasal feed of eggs, milk-and-brandy, and a further hypodermic injection of strychnine. Later in the day she was able to swallow liquids, but at night the nasal feed and strychnine were repeated. In spite of this she continued to be drowsy throughout the following night, requiring more strychnine early in the morning of the second day, but after this she made rapid progress and was able to take plenty of nourishment by the mouth. The stupor gradually left her, and in another twenty-four hours she had completely recovered.

Metaldehyde or Metacetaldehyde, sold under the name of Meta in the form of tablets, is extensively used as a solid fuel. It has caused a number of deaths, especially amongst children, who have eaten the substance in mistake for sweets. The symptoms vary, but usually there is pain in the abdomen, vomiting, convulsions or cramps, and gradually increasing stupor.

For a case of poisoning, see *Analyst*, September 1927, p. 528. Three cases are reported by Leschke (" Clinical Toxicology," 1934, p. 266).

Analysis. Metaldehyde is a white crystalline solid, subliming at 112° C. Paraldehyde is a colourless liquid with a characteristic penetrating odour ; it boils at 124° C. Both, when subjected to distillation from acid solution, form acetaldehyde. This reduces Fehling's solution and ammoniacal silver nitrate, gives a red colour (with green fluorescence) with *m*-phenylene diamine hydro-chloride, etc. (See also, Methyl Alcohol.)

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Poisoning by Chloral Hydrate, $CCl_3CH(OH)_2$

Source and Method of Occurrence. Chloral hydrate is a solid crystalline substance, produced by the reaction of chlorine on ethyl alcohol, and the subsequent addition of a small quantity of water. It has a peculiar hot nauseous smell and taste, which makes it very unsuitable for homicidal poisoning, though it has been feloniously administered in beer. It

has been given in very large doses, often with benefit, but at other times it has caused dangerous symptoms, followed by death. The deaths have frequently been sudden, without remarkable symptoms, the person passing from sleep into death.

Toxicity and Fatal Dose. Chloral hydrate is given therapeutically in doses of 5-20 grains and therefore cannot be considered in itself to have a high degree of toxicity, but in diseased conditions of the heart it is a dangerous drug. Its action seems to be mainly exerted on the cortex of the brain, which is depressed and eventually paralysed. The effect on the medulla is shown by a slowing of the respiration and fall of blood-pressure. The pulse is slowed by direct depression of the cardiac muscle. With large doses the respiratory centre is paralysed. The action on the heart in chloral poisoning is similar to the effect of chloroform. The view has been held that in the blood chloroform is evolved from it, but this is incorrect. Urochloralic acid would seem to be the form in which it is eliminated by the kidneys.

The lethal dose for an adult averages about 150 grains (10 grammes), but varies greatly; three grains in a child a year old are stated to have caused death.[1] Thirty grains caused the death of a woman, aged 30.[2] A dose of thirty grains proved fatal within thirty-five hours to a young lady aged 20. A man recovered from a dose of 160 grains taken at once.[3] Fuller states that one of his patients took 150 grains and another 180 grains without injury.[4] Richardson states that the largest dose which he has known to be taken was 120 grains. It produced prolonged and dangerous coma, but recovery followed. In one instance ninety grains introduced into the rectum produced rapid insensibility, and caused death in three hours.[5] Recovery has taken place after a dose of 240 grains.[6] Even larger doses may be taken by those who habituate themselves to the use of chloral.

There appears to be considerable uncertainty in the action of this drug, even when similar doses are given. After an ordinary dose of twenty or thirty grains a patient has slept for a quarter of an hour, and has then awakened with a sense of deadly faintness, the lips livid, the face pale, the pulse scarcely perceptible, and a feeling of intense exhaustion and impending dissolution, mingled with delirium, lasting for five or ten minutes. Its depressing action on the heart may in cases of heart disease cause rapid death.

Duration. A patient may die within a few minutes, but more commonly survives for a few hours. Death has ensued as late as ten and even thirty-five hours, but in general it is rarely delayed beyond five hours.

Symptoms. In the great majority of cases the only symptom is a quiet sleep which gradually deepens into coma and death, but there may be some gastric irritation and vomiting. Jaundice and albuminuria may occur from the toxic effect on the liver and kidney respectively, breathing becomes gradually slower, till in the end it entirely ceases. The pulse

[1] "Phil. Med. and Surg. Rep.," 1871.
[2] *Lancet*, 1871, 1, p. 226.
[3] *Ibid.*, 1870, 2, p. 402.
[4] *Ibid.*, 1871, 1, p. 403.
[5] *Lancet*, 1871, 2, p. 466.
[6] *B.M.J.*, 1892, 2, p. 1055.

becomes rapid, irregular, and weak. There is complete loss of sensation with great muscular prostration ; the temperature falls much below the normal, and the patient often sweats profusely. It is probable that death usually takes place from paralysis of the heart.

A patient took thirty grains of hydrate of chloral at night. He became unconscious almost immediately after swallowing the draught—the face and hands turned livid and cold, and breathing took place only at long intervals, indeed for about five hours death seemed to be impending. He recovered next day.[1] It has been observed, in reference to this drug, that in the sleep produced by it the pupil is contracted, but that it immediately dilates on the person waking. In other cases the pupil has been dilated and insensible to light.

A *chronic* form of chloral poisoning is known in which the symptoms are mainly digestive, nervous, cardiac, and cutaneous. Pain, nausea, vomiting, and gastric catarrh, due to local action of the drug, are often very striking. In addition a feeble pulse with vaso-motor disturbance, such as redness of the face, may persist and be associated with neuralgia, convulsions, tremor and various psychic phenomena of a depressive character, sometimes ending in melancholia. In severe cases symptoms of delirium tremens may supervene. Erythematous rashes and œdematous patches have been observed. Tolerance is not complete and sudden death may occur.

Treatment. The stomach should be emptied by the tube. Warmth is of the utmost importance ; it should be maintained by hot bottles, and the body should be surrounded by blankets, underneath which friction may be applied. Persistent attempts at rousing the patient should be made by means of the faradic current and other usual methods. If the breathing fails, artificial respiration should be performed, methozol and ephedrine are sometimes of service. Hypodermic injections of strychnine (one-twenty-fifth of a grain) have been recommended, but strychnine is not so good an antidote to chloral hydrate as chloral hydrate is to strychnine. Stimulants will probably be required, ether hypodermically, or alcohol by mouth or rectum. Hot coffee is useful.

Post-mortem Appearances. There is nothing at all characteristic to be found on autopsy. The peculiar odour of chloral hydrate may, however, be perceived in the stomach contents.

Analysis. Chloral hydrate is a white, brittle, crystalline solid, with a peculiar odour and a pungent bitter taste. When heated on platinum it melts, and is entirely volatilised without combustion. It is not inflammable. Heated in a closed tube, it melts, and does not rapidly solidify on cooling. It is distilled over in a liquid form, and after a time it sets into groups of crystals. It is soluble in water. The solution is not acid, has no bleaching properties, and gives only a faint milkiness on boiling with a solution of nitrate of silver. It is dissolved by strong sulphuric and nitric acids, without any change of colour. Potash added to the solution converts it into chloroform and formic acid, which combines with the alkali. On boiling with potash, the solution, if the hydrate is pure, acquires only a slight yellow colour. When boiled with nitrate of silver, and alcoholic solution of soda added, silver is immediately precipitated. It reduces Fehling's solution like grape-sugar.

[1] *Lancet*, 1870, 2, p. 402.

The following delicate test is recommended by Gettler (*Proc. Soc. Exp. Biol. and Med.*, 1919, 16, 110), who claims that it is not affected by other aldehydes, formic acid, or chloroform. To 1 c.c. of the test solution (*e.g.*, steam distillate of stomach contents) add 1 c.c. of saturated resorcinol solution and 1 c.c. of saturated sodium carbonate. Allow to stand for half an hour and add 10 c.c. of water. A green fluorescence (best seen in bright light against a black background) appears if 0·1 mg. or more of chloral is present. Colour is to be ignored.

One hundred parts of chloral will yield eighty-one parts of chloroform, and by this conversion hydrate of chloral may be detected in the contents of the stomach. The liquid should be rendered alkaline with potash, and the mixture heated in a flask on a water-bath. The vapour which escapes may be tested for chloroform by the process described on p. 528. Formates should also be tested for.

The quantity of hydrate of chloral present in a strong solution (*e.g.*, a draught—may be approximately determined by placing a measured quantity of the solution in a graduated and stoppered burette, and shaking with a solution of soda. On allowing the mixture to stand the chloroform formed by the decomposition of the hydrate of chloral will form a dense layer at the bottom. Approximately each minim of chloroform separated represents two grains of hydrate of chloral. By adding a solution of soda of known concentration to a definite volume of a solution of chloral, and, when the chloral is decomposed, titrating the uncombined soda, the percentage of chloral in a dilute solution may be determined. A hundred grains of the hydrate when distilled with lime yield seventy-two grains of chloroform.

A very delicate test for chloral hydrate is its reaction with ammonium sulphide, which is thus described by Luff :—

" If to a solution of chloral hydrate a few drops of ammonium sulphide be added, the mixture either quickly or in a short time, according to the amount of chloral hydrate becomes opalescent, and finally acquires a yellowish or reddish-yellow appearance, an amorphous precipitate finally settling to the bottom of the tube ; at the same time a peculiar odour is developed."

Cases. In 1889 a man was convicted of murder by the administration of hydrate of chloral in beer to an elderly gentleman.[1] Insensibility quickly supervened, and within a few minutes of the victim being found insensible in a cab he died. Chloral was detected in the stomach of the deceased, but no attempt was made to estimate the quantity. In 1891, a man was accused of murdering his wife by the administration of hydrate of chloral ; but he was acquitted, there being no evidence that it was not a case of suicide. Sir Thomas Stevenson found six grains of the hydrate in the stomach and its contents.[2]

On June 1st, 1904, a coroner held an inquest on a spinster of forty-three who had taken an overdose of chloral, quantity unknown. She was found dead after an interval of about twelve hours.

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[1] Manchester Spring Ass., *R.* v. *Pardon*.
[2] *R.* v. *Wynne*, C. C. C., October 1891.

Poisoning by Tribromethyl Alcohol (Avertin)

Avertin is a solid crystalline substance, moderately soluble in hot water, easily soluble in alcohol and ether. It melts at 80° C. It has been regarded as a fairly safe anæsthetic but accidental deaths following its use have been reported.[1] Non-fatal, though alarming cases of poisoning have also occurred.[2]

When its aqueous solution is boiled, avertin is decomposed to hydrobromic acid and dibromacetaldehyde. It should therefore be sought, among the volatile poisons, in the stream distillate from stomach or intestinal contents, viscera, etc. The hydrobromic acid will give a yellowish precipitate with silver nitrate and will yield bromine on addition of chlorine water. The dibromacetaldehyde will give the reactions with Fehling's solution, ammoniacal silver nitrate, Schiff's reagent, etc., characteristic of aldehydes.

Poisoning by Chloralose ($C_8H_{11}Cl_3O_6$)

Source and Method of Occurrence. This drug is formed by the action of chloral on glucose. It is absorbed more slowly than chloral, and has more of a morphia-like action. It also increases the excitability of the reflexes, and may cause strychnine-like convulsions. It is supposed to be much safer as a hypnotic than chloral hydrate, but ill effects have followed its use, especially in large doses. A dose of twenty centigrammes caused very unpleasant symptoms,[3] and eight grains have also done so.[4]

Cases. Dr. G. H. Lang records the following case :—

" I found the patient, a middle-aged woman, lying in bed in a semi-comatose condition. She showed signs of irritation on attempts being made to wake her, but she could not be roused sufficiently to give an intelligible answer to any question. The face was congested and bluish, pupils equal and somewhat dilated, breathing normal, pulse sixty, regular, fairly full, and of high tension, skin warm and moist. On a table by the bed was a box which had contained cachets of chloralose (0·20 grammes in each), but was now empty, and a bottle of syrup of chloral, from which about ℥ vj. were missing. I concluded that she was suffering from poisoning by chloralose or chloral or both.

" As it appeared that the amount taken might be large, I proceeded to wash out the stomach. The water returned almost clear and without smell. I gave an enema of hot coffee. Soon after this she became sufficiently conscious to tell me that she had taken only three cachets, and that she had often taken two, and on more than one occasion three, cachets without ill effects.

" On visiting her later I found her quite well except for a slight headache. She told me that she had taken hypnotics for many years. Once before she had suffered from a very large dose of chloralose, but quickly recovered after an emetic. On the present occasion she had taken two cachets at 1 a.m. and a third about an hour later. I saw her at 4.30 a.m. She thought she slept for a short time after taking the cachets, then felt very ill in an indefinite way, tried to open the door, which was locked, but fell down and was found there in an unconscious condition.

" The chloralose which she was in the habit of taking was made by Bain & Fournier, of Paris, but she had recently obtained a fresh supply from another firm, and two of the cachets taken were from this consignment. The contents of one of them weighed between grs. iij. and grs. iijss., that is, about 0·20 grammes.

" There is nothing in the symptoms to call for remark. They were sufficiently grave to call for active treatment. The quantity taken—0·60 grammes (grs. x.)—

[1] *J. Amer. Med. Assoc.*, 1938, *3*, 122.
[2] *B.M.J.*, 1939, 21st Jan.
[3] *Lancet*, 1895, 2, p. 684.
[4] *Ibid.*, 1930, 2, p. 1803 : *vide* also Professor Bradbury's " Croonian Lecture," 1899.

is the full dose recommended, but she had previously taken as much without ill effects. The untoward symptoms may have been due to rapid absorption from the empty stomach or to impurity in the new supply."

The following appears in the *Lancet*, 2, 1900, p. 1803, reported by Dr. Douty :—

" I was called at about twelve midnight to a young woman who was supposed to be in a fit. She had gone to bed at 11 p.m. She had been in the habit of indulging in chloral and also in cocaine at home, but she had taken very little while at Davos. A physician in London had given her cachets of chloralose, eight grains in each, to take for sleeplessness. She took one for the first time that night. I found her in great distress, unable to move her legs or her arms, with a feeling of numbness in them, the numbness being more marked on the right side. She could not move. Her mind was clear, but her speech was slow. Her pulse was good. She recovered in five or six hours. A week afterwards she took another cachet, and I was called at about 1 a.m. by her friend, who slept in her room, because she would not answer when spoken to and again was thought to be in a fit. She was in a cramped-up posture of emprosthotonos, her thighs were tightly flexed on her abdomen, and her chin was forced down on her chest. Respiration was almost inappreciable and very slow, and she was cyanosed. Her pulse was eighty and fairly good. Her extremities were not cold. She was quite unconscious, and I could not rouse her. I had difficulty in overcoming the contraction of her limbs and neck, but having done so, I performed artificial respiration for half an hour, whilst others applied very hot water to her chest and head, and as soon as she could swallow we forced down some brandy and her breathing improved, but was very slow. In an hour it was possible to get a grunt in reply to questions. She remained in that state for six hours and was torpid all the next day, then seemed quite herself again. The small extent to which the heart seemed affected on each occasion quite bore out Professor Bradbury's observations in his experiments."

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THE SULPHONE HYPNOTICS : SULPHONAL ; TRIONAL (ETHYL SULPHONAL) ; TETRONAL (DIETHYL SULPHONAL)

Poisoning by Sulphonal, Dimethyl-methane-diethyl-sulphone

$$\left(\begin{matrix}CH_3\\CH_3\end{matrix}\right)>C<\left(\begin{matrix}SO_2\cdot C_2H_5\\SO_2\cdot C_2H_5\end{matrix}\right)$$

Source and Method of Occurrence. It is produced by the oxidation of a compound of ethyl mercaptan and acetone. It is official, and its dose is given as ten to thirty grains. It is a pure hypnotic, and has no analgesic action. It is supposed to be a safe hypnotic, but accidents have occurred from its administration, and the habit of taking it may easily be acquired. No case of its homicidal use is yet recorded.

Toxicity and Fatal Dose. In the *Lancet*, 1904, 1, p. 219, a case of recovery after 365 grains is reported, so that its toxicity is slight. Dr. Maidlow reported a case in which " a dipsomaniac, with a mixed motive of suicide and a desire to sleep after an outbreak, twice took 125 grains in ten minutes, the only result being slight stupor and weakness of the legs."

B.M.J., 1895, 1, p. 698, recovery after between five and six grams; *Lancet*, 1909, 1, p. 811, death from twenty grains.

Duration. Sulphonal is apparently very slow in action, owing to the slowness of its absorption, some hours elapsing between the taking of the dose and the onset of sleep. Recovery is also a matter of hours, owing to the slowness of excretion. It tends to accumulate in the blood, and if given regularly, cumulative effects may be produced.

Symptoms. The most prominent symptom is deep and prolonged sleep with gradual failure of the respiration and circulation. In chronic poisoning or acute poisoning in a patient who has been taking the drug for some time there may be acute abdominal pain, vomiting and diarrhœa with nervous depression, confusion and atoxin. The urine may show the presence of hæmatoporphyrin red cells and albumin. Skin eruptions may be seen. It causes a certain amount of destruction of the red corpuscles and seems to have a tendency to upset the renal function.[1]

The following case suggests that sulphonal may seriously affect the kidneys:[2]

"An insane woman, aged seventy, received one gram of sulphonal every evening, the dose having to be doubled in a month's time. This was continued for more than three months, with frequent intermissions, amounting often to eight days. When the total amount taken was some 151 grams the urine showed a dark colour due to hæmatoporphyrin. The drug was omitted at once. Fatal coma, however, supervened in a week's time. The kidneys were of a pale red colour, with smooth surface, and contained deposits of lime salts. These changes were such as might be attributed to advanced age, but the microscope displayed a very extensive necrosis of epithelium, and also minute hæmorrhages. These minute changes represented a toxic nephritis, and were unquestionably due to the sulphonal. During the use of sulphonal the urine should be carefully examined for albumen or formed elements, as when once hæmatoporphyrinuria appears the prognosis is unfavourable. In the presence of renal disease the prolonged use of sulphonal should be had recourse to with great caution."

Treatment. The stomach should be washed out on the chance of removing some of the material not yet absorbed, and a suspension of animal charcoal introduced. Strychnine, hot coffee, etc., may be given and treatment continued on general lines (*vide* pp. 250 *et seq.*).

Post-mortem Appearances. These show nothing diagnostic.

Analysis. Sulphonal is sparingly soluble in cold, moderately in hot, water (1·6 per cent.), soluble in alcohol (1·25), chloroform (33), and ether (0·8). Its melting point is 125° C. If a little dry sulphonal is heated in a test-tube with charcoal, mercaptan is formed along with organic acids and sulphur dioxide; the mercaptan is recognisable by its smell, the SO_2 by its bleaching action on filter paper soaked in starch and iodine solutions. If iron is used instead of charcoal, the subsequent addition of hydrochloric acid to the residue liberates sulphuretted hydrogen. If a little dry sulphonal is melted, and the heat is continued until the clear liquid boils, the addition of pyrogallol produces a brown colour with evolution of mercaptan (Mann). *Vide* also B.P. (1898) tests for the purity of the drug.

[1] "Extra Pharm.," 1904.
[2] *Deut. med. Woch.*, March 8th, 1894; *B.M.J.*, April 1894.

To separate sulphonal from tissues, stomach contents, etc., the material is acidified with HCl and evaporated to dryness on the water-bath. The residue is extracted with chloroform and the extracts evaporated. The residue from this is repeatedly washed with warm petrol ether which removes fats, but leaves the sulphonal. This is then dissolved in water and the solution filtered to remove cholesterol, etc. Extraction of the aqueous solution with chloroform and evaporation of the extract yields a moderately pure residue of sulphonal.

Cases. In 1889 Sir Thomas Stevenson was consulted in a case of sulphonal poisoning, which occurred in the practice of Blatherwick. A lady, *æt.* 40, a dipsomaniac, took a dose of sulphonal, estimated at forty or fifty grains. She was seen twelve hours later, when her condition was as follows: extreme drowsiness, awakened with difficulty, and soon relapsing into somnolence; surface pale and ashen in hue; limbs flaccid, with great loss of muscular power; eyelids closed; pupils moderate in size and acting slowly to light; pulse 120, small; respiration quick, shallow, and at times almost imperceptible: a tongue coated, no vomiting; very offensive odour of body and breath. No urine was passed for twenty-four hours, when it was drawn off, but no sulphonal could be detected in it. The patient was with difficulty made to swallow, and retained fluids in the mouth. She could be made to speak slowly, and with slurring of the words. She recovered in forty-eight hours.

Dillingham has also recorded a case of poisoning by sulphonal. An elderly woman took ninety grains by mistake one evening. At 3 a.m. she was found in a semi-comatose state, which quickly passed into stupor with stertorous breathing. The pulse was almost imperceptible, and the extremities cold. There was well-marked want of control over the muscles, the face was drawn to the right side, and the right eyelid dropped. Pupils normal. She would doze for a few minutes and then open her eyes, but recognise no one. Urine normal. She recovered under treatment, but the partial paralysis of the face and want of control over the muscles continued for ten days.[1]

Lovell Gulland describes a fatal case where the patient had, without the knowledge of his medical adviser, taken thirty grains of sulphonal every night for about six weeks. About a week before his death he developed ataxia of the extremities, lassitude and drowsiness and then hæmatoporphyrinuria, and died somewhat suddenly. The *post-mortem* examination showed no very marked gross lesions beyond fatty degeneration of the heart and stasis in all the organs, but when these were examined microscopically the principal change was found to be necrosis of the secreting epithelium of the kidney, and certain degenerations in the liver and suprarenal capsules.[2]

"Wien[3] relates a fatal case of subacute poisoning in a woman, aged thirty-two, suffering from paranoia. The patient received three doses, each of 0·5 gram, at intervals of an hour and a half, on each of thirty-one days, with occasional intermissions. The urine was examined daily, and the patient had been treated previously with about the same doses with no ill effect. When the sulphonal was discontinued the patient had become quiet, but this was not the result of any sulphonal narcosis. Her general condition was good. Thirty-six hours later the symptoms of sulphonal poisoning appeared. They consisted at first of gastric symptoms, with pain and vomiting, and later paralysis and hæmatoporphyrinuria were noted. It appears probable that the poisoning was due to a cumulative action. Besides the ataxia there was paralysis in the arms and legs, probably due to a peripheral lesion. A notable fact in this case was the late appearance of the hæmatoporphyrinuria, which occurred eight days after the onset of the intoxication symptoms. Albuminuria and other evidence of a toxic nephritis appeared later. The pulse-rate fell towards the end to sixty-eight as a result of changes in the myocardium. At the necropsy a toxic nephritis and cystitis were found. The heart muscle showed degenerative changes, and the pericardial sac contained an excess of fluid. The great danger in sulphonal poisoning lies in the irreparable changes found in the heart. In the stomach

[1] *Med. Record*, December 13th, 1890.
[2] *B.M.J.*, 1898, 2, p. 1821.
[3] *Berl. klin. Woch.*, September 26th, 1898; *B.M.J.*, April (1898), 2 : 332.

there were small haemorrhagic erosions, and the organ presented an hour-glass contraction due to old ulceration. Nine-tenths of the case of fatal sulphonal poisoning have occurred in women. Although the number of cases of poisoning by sulphonal is small considering the frequency with which it is used, yet precautions must be taken. The use of this agent must be as limited as possible, and intermissions of even four or five days are too short. The author thinks that when symptoms of poisoning have already appeared, transfusion, or failing that, infusion of saline solution should be tried. Camphor should be used in case of the least threatening of cardiac failure. Efforts should be made to promote the excretion of the sulphonal by diuresis, etc."

Poisoning by Trional (Diethyl-sulphone-methylethyl-methane)

$$\left(\begin{matrix}C_2H_5\\CH_3\end{matrix}\right)>C<\begin{matrix}SO_2\cdot C_2H_5\\SO_2\cdot C_2H_5\end{matrix}$$

and Tetronal (Diethyl-sulphone-diethyl-methane)

These compounds are more soluble than sulphonal and thus act more quickly, but they are similar in their action to that drug.

"Warren Coleman records in the *Medical News* of July 28th, 1900, a rare and interesting case of acute poisoning by trional. The symptoms of poisoning by this drug have been said to consist of vertigo, loss of equilibration, ataxia, nausea and vomiting, diarrhœa, stertorous breathing and cyanosis, tinnitus aurium, hallucinations, and hæmatoporphyrinuria. The case recorded by Dr. Coleman is that of a woman, aged thirty-five years, who was suffering from a mild delirium brought on by an excessive indulgence in champagne and alcoholic drinks for several days. Six trional powders (of fifteen grains each) were ordered to be taken, one every half-hour for two hours. The patient became drowsy and sleepy, and on the third day it was found difficult to rouse her. There were no disturbances of respiration or circulation to be detected by ordinary clinical examination. In answer to questions she replied that she was 'dizzy and sleepy.' Her speech was thick, and she walked with difficulty, the gait being ataxic. She showed no sensory disturbances, and the urine was free from hæmatoporphyrin. Upon inquiry it appeared that she had taken trional far in excess of the prescription and to the extent of nine drachms (540 grains) in seventy-two hours, the original prescription having been repeatedly made up at the stores. The patient was ordered a saline purge, and she gradually recovered from her state of trional intoxication. The above case shows that trional is free from excessive depressing effects even when taken in maximal medicinal doses, and that even when by accident enormous doses are taken the result need not be necessarily fatal. The development of hæmatoporphyrinuria should always be looked upon as a danger signal in cases of administration of trional or sulphonal, and the use of the drug should be discontinued until this symptom has passed away."

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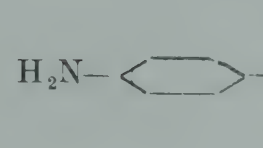
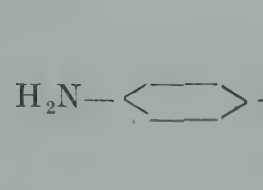
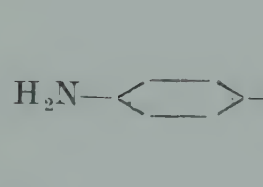
Poisoning by Sulphonamides

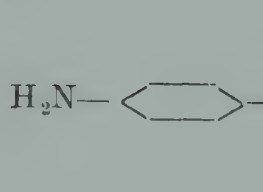
Source and Method of Occurrence. The sulphonamide drugs, of which a great many are marketed for the treatment of various systemic bacterial infections, are derivatives of *p*-amino-phenyl-sulphonamide, $H_2N-\langle\ \rangle-SO_2\cdot NH_2$. There are two main series: one in which a hydrogen of the sulphonamide group is replaced by a radicle, the resulting compound being still a primary amine; the second in which a substituting radicle replaces a hydrogen of the amino-group to give a compound which may or may not be a primary amine. The following list of the most commonly used drugs of these series gives the names under which they are usually sold and their melting points.

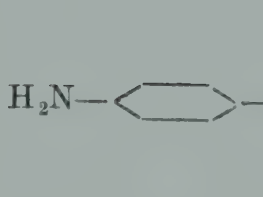
Sulphanilamide. m.p. 165–166° C.


Sulphapyridine, Dagenan, M and B 693. m.p. 191–193 °C.

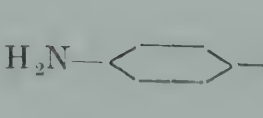

Sulphathiazole, Thiazamide, Cibazol. m.p.202–203° C.


Sulphamethylthiazole, Ultraseptyl. m.p. 237–238° C.


Sulphadiazine, Pyrimal. m.p. 255–256° C.


Sulphamezathine m.p. 197° C.

Sulphacetamide, Albucid m.p. 183–184° C.

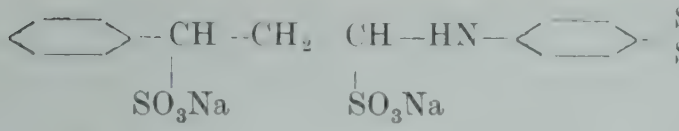

Sulphaguanidine m.p. 189-190° C.

Diseptal C., Uleron C.

Diseptal A, Uleron. m.p. 194° C.

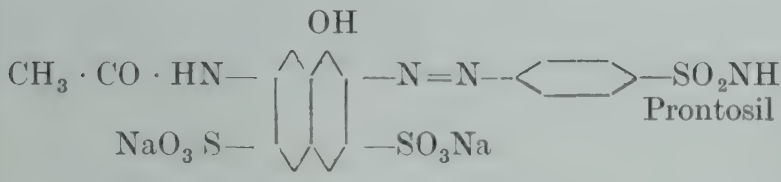
II.

Septasine, Proseptasine m.p. 179–181° C.

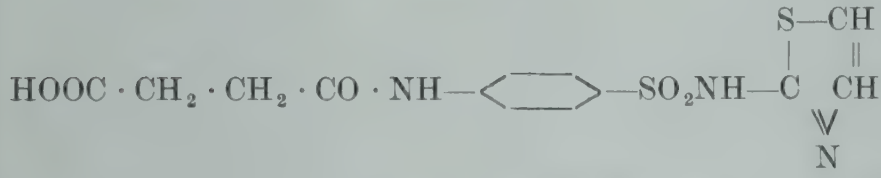


SO_2NH_2
Soluseptasine,
Solucin.

Prontosil, Prontosil morum. m.p. 248-250° C.



Prontosil soluble.



Succinylsulphathiazole. m.p. 183–185° C.

In addition to the true sulphanilamides, derivatives of 4·4′-diamino-diphenyl-sulphone, $H_2N—C_6H_4—SO_2—C_6H_4—NH_2$ are employed as bacteriostatic or bactericidal agents as are substances derived from homologues of sulphonilec acid (*e g.*, Marfanil, $H_2N \cdot CH_2—C_6H_4—SO_2 \cdot NH_2$).

Although, in general, the sulphonamide drugs have a satisfactory margin of safety, mild toxic symptoms are not uncommon after therapeutic doses, and in a small but appreciable number of cases there are more dangerous effects and deaths have been reported.

Toxicity and Fatal Dose. No general statement is possible, for not only do the sulphonamide drugs differ in toxicity when tested experimentally in animals, but the susceptibility of individuals (animals and man) for each drug, varies over a wide range. Doses used therapeutically with benefit to many patients have had serious and even fatal effects in a few cases. On the whole, it is fair to say that sulphapyridine is more toxic than sulphathiazole, which in turn is more toxic than sulphanilamide.

Symptoms. Mild toxic symptoms which are by no means uncommon after administration of sulphonamides include dizziness, headache, nausea, vomiting, diarrhœa, pyrexia, cyanosis, skin rash, etc. Thus nausea is described as "common" after sulphapyridine, though rare after sulphathiazole; cyanosis is common after sulphanilamide or sulphapyridine, uncommon after sulphathiazole; fever occurs in about 10 per cent. of patients receiving sulphanilamide or sulphathiazole and in 4 per cent. of those given sulphaipyrdine.

The more serious toxic actions of the sulphonamides are far less common. They are on the skin and, more importantly, on the bone-marrow, blood, and kidneys, though hepatitis has also been reported. Acute hæmolytic anæmia has been found as the result of medication with both sulphanilamide[1] and sulphapyridine[2] and fatal cases have been described. Severe neutropenia, occasionally resulting in a fatal agranulocytosis,[3] has been reported more frequently. Renal damage may result from precipitation in the renal tubules, renal pelvis, or ureter, of sparingly soluble acetyl-derivatives of the sulphonamides, and be shown by hæmaturia, depression of renal function with nitrogen retention, formation of calculi, and even complete anuria; fatal cases have been reported,[4] chiefly after the use of sulphapyridine. Pre-existing renal damage, by slowing the excretion of the drugs, may increase their concentration in the blood, and consequently their toxic effects.

Treatment. Treatment will, naturally, be directed to the type of toxic reaction present. Usually it will include witholding the toxic agent, the sulphonamide drug. It must be remembered, however, that in the treatment of serious or desperate illness, the physician may have to weigh the relative risks of witholding treatment and of continuing the sulphonamide drug even in the presence of toxic symptons. Thus, though microscopic hæmaturia is an absolute indication for stopping the administration of sulphapyridine, etc., continuance may be justified even in the presence of microscopic hæmaturia. Similarly, a dangerous infection may justify continued administration of sulphonamides in the face of a hæmolytic anæmia, when it is possible to replace the destroyed red cells by blood transfusion at suitable intervals.

Post-mortem Appearances. The appearances to be expected post-mortem are those appropriate to the toxic effects seen ante-mortem. With renal symptoms it is important to search for uroliths or calculi which can be chemically identified as consisting of the acetyl derivative of the sulphonamide concerned.

Analysis. Complete identification of a sulphonamide present in organic material—blood, urine, stomach contents or tissues—is not easy for the colour reactions available are not specific, and it becomes necessary to isolate the substance in a pure state and to examine its physical properties—crystalline shape, melting point, etc.—in comparison with pure samples. For extraction, the most suitable method is to dry the material, first by evaporation and then by grinding the residue with anhydrous sodium sulphate, and to extract this with alcohol in a Soxhlet extraction apparatus. Since the drug may be present in part as the acetyl derivative, a preliminary hydrolysis by boiling with dilute hydrochloric acid for thirty minutes is desirable.

[1] Wood, *South. Med. J.*, 1938, *31*, 646.

[2] Long & Wood, *Ann. Int. Med.*, 1939, *13*, 487; McLeod, *J. Amer. Med. Assoc.*, 1934, *113*, 1405.

[3] Long & Bliss, *J. Amer. Med. Assoc.*, 1939, *112*, 115; 1940, *115*, 364. Shecker & Price, *ibid.*, 1939, *112*, 823.

[4] Smith & Needles, *Amer. J. Med. Sci.*, 1939, *198*, 19. Tsao, *et al.*, *J. Amer. Med. Assoc.*, 1939, *113*, 1316. Finland, *et al.*, *J. Clin. Invest.*, 1940, *19*, 179.

When, as is usually the case, it is merely necessary to *confirm* the presence of a sulphonamide drug known from the case history to have been administered, the qualitative tests and possibly a quantitative determination, will usually suffice.

The Azo Dye Test. To about 20 mg. of the substance dissolved in about 2 ml. of dilute hydrochloric acid, add 2 ml. of approx. 2·5 per cent. sodium nitrite solution. After two or three minutes add a few drops of an alkaline solution of β-naphthol. Sulphonamides (and other arylamine compounds) with a free primary amine group give a red or orange colour. Proseptosine which is not an amine, does not react, but in the body it is partly broken down to sulphanilamide (which does give the test) and this substance is excreted in the urine.

The p-Dimethylaminobenzaldehyde Test. This is given by those sulphonamides which are primary amines (and by other aromatic amines). To a few ml. of aqueous solution containing about a mg. of the drug, add about 1 ml. of a 3 per cent. solution of *p*-dimethylaminobenzaldehyde in 7 per cent. sulphuric acid. A yellow colour develops immediately, and, if the concentration is high, an orange precipitate.

Both of these reactions have been made the basis of quantitative methods, the azo dye test by, *e.g.*, Bratten and Marshall[1] and the dimethylaminobenzaldehyde test by Werner.[2]

Modified Method of Bratton and Marshall. This depends on the production of an azo dye by diazotising and coupling with a suitable amine. It can be used for whole blood, plasma, cerebrospinal fluid, urine, etc. Blood should be well oxalated and free from clots, which adsorbs the sulphonamides.

Reagents :

p-toluene sulphonic acid, 16·7 per cent. (w/v).

Sodium nitrite, 0·1 per cent. (w/v) made fresh every fortnight.

Urea, 0·5 g. and sodium dihydrogen phosphate 6·9 g. in 100 ml. distilled water.

Dimethyl-naphthylamine, 0·4 g. in 100 ml. of 97 per cent. alcohol.

Standard solution : prepared by dilution from a stock solution containing 20 mg. of the appropriate sulphonamide per 100 ml. dissolved with the aid of sodium hydroxide.

[It is possible to use a single standard of, say, sulphapyridine, using the appropriate factor for the determination of other members of the group. With a sulphapyridine standard the factors are : sulphanilamide 0·68, sulphathiazole 1·04, sulphamethylthiazole 1·11, sulphadiazine 1·0].

Take 2 ml. of blood with an equal volume of water in a 25 ml. graduated flask, add 4 ml. of the toluene-sulphonic acid solution, mix, make up to the mark with water, stand five minutes and filter. Similar quantities of plasma or C.S.F. are treated similarly except that there is, of course, no laking. Urine is best diluted to give a solution containing about 5 mg. sulphonamide per 100 ml. and then treated as plasma. (The colour with urine is, however, less satisfactory than with blood.)

[1] *J. Biol. Chem.*, 1939, *128*, 537.
[2] *Lancet*, 1939, *1*, 18.

To determine " total " sulphonamide, heat 10 ml. of the filtrate on the boiling water-bath for an hour, cool, and make up to the original volume before proceeding further. For " free " sulphonamide, to 10 ml. of filtrate (unboiled), add 1 ml. of the nitrite solution, mix and stand 3 minutes. Add 1 ml. of the buffered urea solution to destroy excess nitrite and, after standing 3 minutes add 5 ml. of the dimethyl naphthylamine. Compare the colour with that of a standard similarly treated.

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Church, " Trional Fatalities." *Amer. Med.*, 1901, 2, 729.
McIntosh, *Lancet*, 1901, 1, 104.
Rolleston, *Trans. of Clin. Soc.*, 1896, 30, 125.
Wightwick and Rolleston, *Lancet*, 1903, 1, 1096.

Poisoning by Bromoform ($CHBr_3$)

Source and Method of Occurrence. Bromoform is the bromine analogue of chloroform ; it is a colourless, sweet liquid, with a great reputation as a sedative in whooping-cough, but has to be given with care. It is only slightly soluble in water, and hence not infrequently the last dose in a bottle contains a poisonous amount.[1, 2, 3, 4]

The following case is typical.[2]

In the early part of last year my partner's five children were suffering from pertussis and, other remedies failing, he decided to try bromoform, one and a half drachms of which were put in a mixture made up to eight ounces with mucilage and water. The eldest girl, aged eight years, took one tablespoonful, and the others smaller doses according to their ages. For four or five days they took the medicine, and were so benefited by it that it was discontinued. On the sixth day, about 4.30 p.m., I was sent for, as my partner was out, and on arriving I found the eldest girl in the following critical condition. She was lying in a wholly unconscious state, snoring deeply. Both pupils were strongly contracted, and hardly reacted to light. The conjunctival reflexes were almost lost, the pulse at the wrist was scarcely perceptible, and the heart-sounds were very weak. The face was pinched, and the lips were blue. I gave two drachms of ipecacuanha wine, which produced fairly free vomiting, and followed this up by a hypodermic injection of $\frac{1}{120}$th of a grain of strychnine, and applied hot flannels over the heart. As the improvement in the child's condition was very slight, I gave an injection of two drachms of whisky under the skin of the chest and an enema of strong coffee. The pupils were now more dilated, and the pulse was better in strength and quality ; and though still unconscious, the patient's general condition had improved. Her father then returned and drew my attention to a peculiar sweetish smell which was evolved from the vomit and breath, and on inquiry we received the following particulars of the onset from the mother. The girl had a violent fit of coughing at 3 p.m., and was given a dose of the bromoform mixture, *the last in the bottle*. She then lay down. From half to three-quarters of an hour later her mother heard loud snoring issuing from the room and, being unable to rouse the child, sent for me About 8 p.m. the patient recognised her father and seemed to be improving. At 10 p.m. there was a change for the worse ; the character of the respiration, however, changed from the loud snoring to a soft sighing, becoming so feeble that we resorted to artificial respiration. Owing to the recurrence of the symptoms, we suspected that some of the drug must be left in the stomach, which we accordingly washed out with an ordinary syphon pump, and noticed that the water which returned had the same sweetish smell, which smell the child's breath, retained for several days. The remaining treatment through the night consisted in putting the patient near an open window, where she gradually recovered, and, with the exception of a very bad headache the next day, she did not seem much the worse for her misadventure.

[1] *Lancet*, 2, 1816 (1898).
[2] *Lancet*, 1 : 119 (1899).
[3] *B.M.J.*, 1 : 1283 (1900).
[4] *B.M.J.*, 1 : 1202 (1901).

The substance cannot be looked upon as very poisonous, though the doses recorded are all very small.

Analysis. If necessary, bromoform can be tested by similar methods to those used for chloroform (*q.v.*, p. 528). Heating, or refluxing with KOH will, of course, yield HBr or KBr instead of HCl or KCl.

THE BARBITURIC ACID GROUP OF HYPNOTICS

When urea is combined with malonic acid a substance known as malonyl urea or Barbituric Acid is formed. Itself an inert body, it readily forms various alkyl and aromatic derivatives to produce a series of hypnotic drugs.

The oldest of these, di-ethyl barbituric acid, was introduced into medicine in 1903 by Fischer and von Mering, under the name of Veronal, and proved itself an effective hypnotic without serious side actions. The structure of the drug lends itself readily to chemical combinations and it is theoretically possible to produce more than 1,200 barbitone derivatives. Great numbers of these substances have been produced and are obtainable commercially. The commonest members of the group are listed in the following table :—

Usual or Official Name	Chemical Structure	Synonyms	Melting Point
1. Allonal	Isopropyl-allyl-barbituric acid + pyramidone.	—	138–141°
2. Amytal	Isoamylethyl barbituric acid	—	155–157°
3. Barbitone	Diethyl barbituric acid ...	Veronal, Barbital, Malonal, Hypnogen, Quadrinox.	191°
4. Barbitone—soluble ...	Sodium salt of barbitone ...	Veronal-Sodium, Medinal	—
5. Barbitone and Amidopyrine	—	Veramon, Veropyron ...	—
6. Buto Barbital ...	Ethyl butyl barbituric acid...	Neonal, Soneryl	123–124°
7. Dial	Diallyl barbituric acid ...	Curral (Dial + pyramidon = Cibalgin)	179°
8. Heboral	Free acid of ortal-sodium ...	—	126°
9. Hexobarbitone ...	N - methyl - cyclohexenyl methyl barbituric acid.	Evipan, Evipal, Cyclonal, Hexanostab-oral.	144–145°
10. Hexobarbitone-soluble	Sodium salt of Hexobarbitone	Evipan-soluble, Cyclonal-sodium, Hexanostab.	—
11. Ipral	Ethyl isopropyl barbituric acid.	—	198°
12. Noctal	Isopropyl - bromallyl barbituric acid.	Nostal	176–177°
13. Ortal sodium	Sodium ethyl - n - hexyl - barbiturate.	—	—
14. Phanodorm	Cyclohexenyl ethyl barbituric acid.	—	178°
15. Phemitone	N-methyl, ethyl phenyl barbituric acid.	Prominal	170–172°
16. Phenobarbitone ...	Ethyl phenyl barbituric acid	Gardenal, Luminal, Somonal, Phenobarbital.	170–172°
17. Phenobarbitone and Theobromine	—	Theoba, Theogardonal, Theominal.	—
18. Pentobarbital ...	Sodium ethyl - methylbutyl - barbiturate.	Nembutal	130–132°
19. Pentothal-Sodium ...	Sodium ethyl-methylbutyl-thio-barbiturate	—	155–157° (free acid).
20. Pernoctal	Isobutyl-bromallyl-barbituric acid.	Pernoston	130–132°
21. Sandoptal	Isobutyl-allyl-barbituric acid	—	138–141°
22. Proponal	Dipsopyl-barbituric acid ...	—	145°

The action of these various derivatives is similar. They are all hynotics, but they vary in their rapidity and intensity of action and their toxicity. A good deal depends on the rate of absorption, the rapidity of destruction in the tissues and on the rate of excretion from the body.

Since barbituric acid itself is inert, the rate of oxidation of the side chains determines the duration of action. Barbitone is very resistant to oxidation, is excreted mainly as barbitone in the urine and its excretion is a slow process. It is, therefore, liable to act as a cumulative poison.

Phenobarbitone is more powerful, but is more easily oxidised and is excreted more rapidly, hence there is less danger of cumulative effects. Nembutal and evipan are broken down more rapidly still, and therefore more rapidly excreted. In general the shortest acting barbiturates are those most easily destroyed. The excretion also appears to be related to the rate of oxidation of the drug, hence barbitone which is excreted almost unchanged, takes longest to excrete. Excretion tends to become slower with continued administration and is definitely more prolonged in nephritic conditions.

Sollman[1] indicates the relative toxicity of certain typical members of the group by the rat lethal dose in mgm. per kg. (hypodermically) as follows:—

Barbitone (Veronal)	310
Phanodorm	220
Neonal	190
Dial	150
Phenobarbitone (Luminal)	140
Amytal	140
Pernoston (Pernoctal)	126
Pentobarbital (Nembutal)	120
Ipral	110

Barbiturates must be classed as habit-forming drugs and as such are potentially dangerous, although it must be admitted that long continued administration under medical supervision has not been followed by bad effects in numbers of cases. These drugs are now under control, their sale and use being restricted under the Poisons and Pharmacy Act (1933), but they still account for a good many suicides each year and also for many accidental deaths.

It has been pointed out that many cases which might be classed as suicidal are really accidental and due to the fact that a dose taken as a hypnotic may cause amnesia and that the patient may continue to take further tablets until a fatal dose has been ingested. It is advisable therefore to make sure that a patient under medication by barbiturates has access to one dose only at a time.[2]

Barbituric Acid derivatives are normally soluble with difficulty in water and are therefore absorbed slowly, but their sodium salts are usually readily soluble and are absorbed rapidly. When used as pure hypnotics, they are usually given by the mouth, but when rapid effect is required the soluble salts may be given intravenously.

The barbiturates are used in a variety of conditions such as for sleeplessness, for excited and anxiety states and other psychiatric conditions, for epilepsy, for convulsions of central origin or the convulsions of strychnine, coramine, picrotoxin or cocaine poisoning.

They are used as general anæsthetics for very short operations or for premedication before ether or similar anæsthesia and for obstetric amnesia and as basal anæsthetics. Their use intravenously requires great care for, once administered, they are beyond the control of the anæsthetist and there is a definite danger of paralysis of the respiratory centre.

[1] *Sollman, Pharmacology*, W. B. Saunders & Co., 1944, p. 786.
[2] Richards, R., *B.M.J.*, 1, 331 (1934).

Acute Symptoms. Amnesia is an early symptom and this may result in the patient taking more tablets than he intends.

The outstanding symptom of barbiturate poisoning is deep sleep passing into coma which is profound and prolonged. There may, however, be a state of excitement and hallucinations before narcosis occurs. The duration of the coma varies from a few hours to several days and is more prolonged with barbitone than with other members of the group. The pupils are sometimes dilated but when coma occurs they may be contracted and unequal. Nystagmus and diplopia may occur.

The temperature is lowered in the early stages but soon rises and may reach high levels, the rise being usually associated with the onset of broncho-pneumonia. The action of the heart is depressed and there is a marked fall in blood-pressure with toxic doses. The respirations are slow, deep and stertorous and in grave cases may be of the Cheyne-Stokes type. Death from respiratory paralysis may occur.

The face is congested, at first red in colour, but becomes more and more cyanotic as the respiratory centre becomes involved. The urine may be diminished or complete anuria may occur. It may contain hæmatoporphyrin, blood or albumen. Incontinence has been observed. The emptying time of the stomach is delayed. The muscles are flaccid. The reflexes are greatly diminished and there may be tremors or spasms. Babinski's sign may be observed. Skin eruptions occasionally occur and jaundice has been reported in connection with severe cases. If the coma continues, involvement of the lung is very liable to occur.

Chronic Symptoms. There is little real tolerance to these drugs and long continued use may lead to a certain degree of psychical degeneration and minor grades of mental aberration. There may be ataxia vertigo, tremor of the hands, paralysis, visual hallucinations, loss of memory, and stammering speech. Cutaneous affections occur. In general, however, it is remarkable how little effect is produced by long continued use of these drugs in large numbers of cases as for example in the treatment of epileptics and the chronic effects described above are only rarely seen. Acute toxic effects on the other hand are not uncommon in habitual users of the drug.

Toxic Dose. The official dose of barbitone is 5 to 10 grains. The medicinal dose of medinal is given as 5 to 10 grains, proponal 2 to 8 grains, luminal $\frac{1}{2}$ to 2 grains.

The fatal dose of the barbiturates is difficult to define with any degree of precision. The long period of coma gives a good margin of time for treatment, and recovery may readily occur after toxic doses if treatment is begun early and is sufficiently intense.

Leschke[1] considers 150 grains of barbitone as a fatal dose in general conditions but records fatalities after 60, 75, 90 and 105 grains and of recovery after 120 grains. An average fatal dose of 60 grains is suggested for Luminal (phenobarbitone) and 40 grains for Dial.

Hambourger[2] gives figures for minimum fatal doses and maximum doses from which recovery has taken place. The lowest fatal dose of barbitone which he reported was 30 grains and of phenobarbitone

[1] Leschke, E., *Clinical Toxicology*; J. & A. Churchill, 1934.
[2] Hambourger, W. E., *J. A.M. A.*, 114 : 2015 (1940); 112 : 1340 (1939).

25 grains. Recovery was reported after 500 grains of barbitone, 140 grains of phenobarbitone and 36 grains of Dial.

Sexton, D. L., *et alia*[1] record a case in which death occurred after a total dose of 42 grains phenobarbitone, half subcutaneously and half orally, over a period of 13 days, that is, about an average of $3\frac{1}{4}$ grains per day.

The toxic dose of veronal ranges between 15 and 150 grains, but a case is on record in which 360 grains were recovered from. This case[2] is rather instructive, and the details are given on pp. 579-80.

A case is reported of death from 100 grains.[3] Carlill[4] records a case in which a woman was given 10 grains luminal and 50 grains potassium bromide per day for twelve days. She became stuperose with double incontinence, pupils contracted and inactive with divergent squint. The reflexes were unaffected. She eventually recovered completely.

Nichol[5] records a case of coma for twelve hours and complete recovery after taking 18 grains of dial.

Leschke[6] mentions six cases of suicide by means of dial, in which the dose was 2 to 2·4 grams, but states that in other cases doses of 6 grams have been survived without permanent damage. He also states that in ten fatal cases of luminal poisoning the average dose was 4 grams, whereas, in one case 6 grams were survived (the victim recovered after being unconscious for a week). Nembutal has been stated to cause death in doses of $7\frac{1}{2}$ and 6·7 grains when used as an anæsthetic in patients acutely ill.

Treatment. The patient is usually comatose and if so, no attempt should be made to produce vomiting but the stomach should be thoroughly washed by successive quantities of warm water and potassium permanganate, leaving a suspension of animal charcoal after the lavage is completed to absorb any poison not removed.

Intravenous injection of normal saline should be given with glucose to assist elimination. Artificial respiration and the use of oxygen and carbon dioxide may be required if the respiration is difficult.

The urine may have to be removed by catheter and bowel evacuation secured by enemata.

The possibility of lung involvement should be kept in mind and appropriate treatment adopted. It is desirable to shorten the period of coma to prevent this and other complications and many analeptics have been experimentally investigated, particularly strychnine, cocaine and picrotoxin, metrozol, cardiozol and coramine. Both strychnine and picrotoxin are valuable but it appears to be established that picrotoxin is the drug of choice in severe cases. Picrotoxin is itself a dangerous drug (dose $\frac{1}{2}$-2 mgm.) and must be used with great discrimination. Overdosage must be avoided and it must not be allowed to produce convulsions which in themselves would increase the depression. It is administered in divided doses of 0·1 mgm. intravenously at intervals of 20 minutes to an hour, until an increase of respirations and a rise in blood-pressure occurs or a slight twitching of the facial muscles is observed. The dosage is then decreased.

[1] Sexton, D. L., *et alia*, *J. A.M. A.*, 116 : 700 (1941).
[2] Tardieu, *Bull. Soc. de Ther.*, February 13th, 1924.
[3] *Lancet*, 1907, 1, 1496.
[4] *Ibid.*, September 19th, 1925.
[5] *B.M.J.*, August 16th, 1924.
[6] "Clinical Toxicology," 1934, pp. 186, 187.

In lethal doses the shorter acting barbiturates promptly produce a state of profound narcosis with a high degree of anæsthesia and cause death from respiratory paralysis whereas in similar circumstances the longer acting members (barbitone, phenobarbitone, dial) produce a low grade of narcosis and slight degree of anæsthesia, but the narcosis is prolonged, ending in pulmonary congestion and other complications. This difference in action of the two groups necessitates a difference in treatment with antidotes such as picrotoxin. The shorter acting derivatives cause rapid paralysis of the respiratory centre and prompt fall of blood-pressure which must be counteracted by pushing the picrotoxin treatment to the physiological limit. In the case of the longer acting derivatives the antidote must be given in smaller and more frequent doses.[1]

A case in which a person who took 72 grains of sodium amytal is described by Stephens and Anderson.[2] The patient was comatose with marked signs of anoxemia, cyanosis, depressed respiration and clonic convulsions. 156 mgm. picrotoxin was given in divided doses over a period of 24 hours together with large quantities of saline intravenously. The patient regained consciousness and recovered rapidly. Metrazol (2 cc. of 10 per cent. solution), Coramine and strychnine ($\frac{1}{2}$ gr. in 24 hours) have also been used with advantage.

Recovery after the ingestion of 125 grains of a mixture of pentobarbitone and seconal has been recorded. The patient was in a state of shock, the respirations were 8 per minute, the blood-pressure 60/0. Shock therapy and picrotoxin injection was instituted and after 20 hours improvement commenced. Delirium and confusion lasted for 24 hours and gradually subsided. There were no convulsions, no lung complications and no evidence of liver or kidney damage. Recovery was complete.[3]

Strychnine in large doses has been found of great value by many observers.[4] Elimination should be assisted by injections of normal saline. The respiration must be watched and artificial respiration resorted to if necessary, but the slow elimination of the drug renders this of little value. Coramin 15 per cent. solution in 5 c.c. doses and also Metrazol has been recommended.

Willcox considers that removal of cerebro-spinal fluid is essential.[5]

Analysis. There are so many barbituric acid derivatives in common use, and their chemical differences are so slight that complete identification is often a matter of great difficulty. It must usually depend on isolation and determination of physical properties such as melting point. Frequently proof of the presence of a barbiturate is all that is possible.

Barbituric acid derivatives in general give :—

(1) Ammonia when heated dry with sodium carbonate.

(2) A white gelatinous precipitate with Millon's reagent.

(3) A violet colour with an alcoholic solution of cobalt nitrate in presence of ammonia.

A modification of this last test—a colour with cobalt acetate and *iso-*

[1] Maloney, A. H. and Tatum, A. L., *Journ. Phar. and Exp. Therap.*, 44 : 337 (1932).
[2] Stephens and Anderson, *Ohio State Med. Jour.*, Columbus, 35 : 396 (1939).
[3] France, C. J., and others, *J. A.M. A.*, 122 : 173 (1943).
[4] *B.M.J.*, December 16th, 1933.
[5] Willcox, W.M, *B.M.J.*, 1 : 493 (1933).

propylamine in chloroform—has been developed by Levvy[1] into a quantitative method of estimation.

For various colour reactions which can be used to distinguish individual barbiturates *see* Bamford, "Poisons, Their Isolation and Identification."

A considerable part of many ingested barbituric acid derivatives is excreted unchanged in the urine, but the individual compounds vary a great deal with respect both to the amount excreted and the rate. Thus it is said that 70 per cent. of veronal escapes oxidation and is excreted, but only 30 per cent. of curral, 25 per cent. or less of luminal, and 5 per cent. of phanodorm. In experiments by Shoule and others,[2] no amytol or pentobarbitone was found in the urine after intravenous injection of their sodium salts in dosages sufficient to cause immediate loss of consciousness. On the other hand, barbitone and phenobarbitone were readily detected in the urine after such administration. It is probable that the former compounds are destroyed in the body. As to the rate of excretion, veronal appears to be excreted much more slowly than the others. Pinkhof[3] finds that the time required for the excretion of half the ingested amount is 13–15 hours in the case of veronal, 7·5 hours for luminal, 9 hours for somnifen, and 4 hours for dial or neodorm.

The urine is thus of importance in the detection of the actual poison used, but no delay should take place in the analysis.

Isolation from Tissues. In the Stas–Otto process (p. 265) the barbituric acids may be extracted from the acid solution by ether. The residue from this extract is dissolved in hot water, any insoluble matter is filtered off, and the filtrate (concentrated if necessary) is re-extracted with ether. The ethereal extract, on evaporation, deposits the crystalline barbituric acid.

In the case of urine, precipitation of interfering substances with lead acetate, removal of excess lead by H_2S, and concentration *in vacuo* forms a suitable treatment preparatory to extraction with ether.

Ethyl acetate has been recommended as a solvent in place of ether, and it has been suggested to purify the extract by acid solution of potassium permanganate.

Tests. *Melting Point.* Wherever possible a melting point of the extract should be taken and followed by the melting point of the extracted substance mixed with an equal amount of the pure drug.

The table on p. 573 gives the melting point of the more important barbituric acid derivatives.

Veronal is a colourless, crystalline solid, which melts at 191° C. Viewed under a lower power ($\frac{2}{3}$), the crystals appear as hexagonal prisms, or in kindred forms. They dissolve with difficulty in water, floating obstinately at the top of the liquid in spite of shaking. The addition of a drop of caustic potash makes solution easy. In the solution so obtained mercurous nitrate solution produces an abundant white precipitate. When a mixture of the dry substance with dry sodium carbonate is heated in a test-tube, ammoniacal vapours are freely evolved which colour

[1] *Biochem. J.*, 1940, *34*, 73.
[2] Shoule, H. A., *et alia*, *Jour. Pharm. and Exp. Therap.*, 49 : 393 (1933).
[3] *Nedel. Tijdschr. v. Genesk.*, 1930, 74, 2189.

moist red litmus paper blue, and moist turmeric paper brown, when these test papers are held in the mouth of the tube (Hugh Candy). (These tests are given by all barbituric acid derivatives.)

Differentiation of Veronal, Proponal and Luminal.[1] In a small crucible melt about 1 gr. of caustic alkali and sprinkle thereon a little powdered veronal: a sour rancid odour becomes immediately apparent. Similar treatment with luminal yields a pleasant odour like locust honey, which later becomes more pungent; with proponal the odour is at first spicy, then becomes pungent. A general colour test for veronal, luminal and proponal consists in dissolving about 0·01 to 0·02 gr. of the sample with a drop of salicyl aldehyde in 1 to 2 c.c. of alcohol, then cautiously adding concentrated H_2SO_4, whereupon the zone of contact becomes intensely red. The following colour test differentiates luminal from both veronal and proponal. To 0·01 to 0·02 gr. of the sample add 0·5 to 1 c.c. formalin and 4 c.c. concentrated H_2SO_4, whereupon the mixture at room temperature gradually, on the steam bath within one minute, acquires a brilliant rose-red, then cyclamen-red to wine-red colour with luminal. With 0·05 gr. of luminal the colour becomes dark mulberry-red. With veronal and proponal only a yellowish colour develops on the steam bath.[2]

Cases. A. Tardieu and S. Camps[3] record a case of acute suicidal poisoning by veronal in a Russian ex-airman, aged thirty, who had taken 24 grammes in a single dose. After taking them he sent himself to sleep by ether. When first seen by the authors, on the fourth day after ingestion of the veronal, he was still in a state of profound coma, the limbs were inert, perspiration was profuse, the face cyanosed, and the pulse barely perceptible. There were incontinence of urine and faeces, loss of the tendon and corneal reflexes, dulling of general sensibility, mydriasis, and profuse secretion of saliva. After the coma had lasted five and a half days the patient slowly regained consciousness. The temperature was of a continued type, sometimes being as high as 102° F. On the seventh day, after an apyrexial interval of forty-eight hours, there was a transient rise of the evening temperature to 100·2°. Examination of the lungs did not reveal any evidence of tuberculosis, pulmonary œdema, or even hypostatic congestion, which is so frequent in acute intoxications. As soon as the patient recovered consciouness he complained of seeing double. On examination he was found to have crossed diplopia, which was very marked for thirty-six hours and then progressively diminished, completely disappearing on the ninth day. There was also slight bilateral ptosis. On examination of the heart signs of old mitral disease were found. In spite of repeated examination of the urine no albumen or sugar was detected. Examination of the urine for veronal on the seventh day was also negative. A psychiatric study of the patient showed the association of two psychopathic conditions—namely, manic-depressive psychosis and paranoia. For the last three years he had been addicted to drug taking, especially hashish and ether. According to Mantelin, Martin, and Milhaud, the toxic dose of veronal ranges between 1 and 11 grams. The fact that so large a dose as 24 grams was not fatal in the present case, in spite of old mitral disease, is explained by the circumstance that, according to Bachem, after consumption of large doses of veronal 45 per cent. of the drug is destroyed without acting on the organism. The presence of ether also probably interfered with the assimilation of veronal.[4]

Dr. Durrant supplied the following details of a fatal case of veronal poisoning, on which an inquest was held in July, 1909.

The victim was a man *æt.* 33. He had been drinking for years, often heavily; veronal had been previously prescribed for him in fifteen-grain doses, also chloral

[1] L. Ekkert, *Pharm. Zentral-halle*, 1926, 67, 481-2.
[2] *Chemical Abstracts*, October 20th, 1926, p. 3330.
[3] *Bull. Soc. de Thér.*, February 13th, 1924, p. 63.
[4] *B.M.J.*, Epit., June 21st, 1924.

hydrate gr. x. and Am. Brom. gr. xx. He had gradually increased the dose of veronal and was taking indiscriminately thirty to sixty grains at times. Was worried by domestic trouble, and often threatened suicide. Had taken 100 grains of veronal with suicidal intent five weeks before his death and recovered in three days. On June 29th, 1909, obtained " two bottles " of veronal in five-grain tablets, retired to bed 12.30 to 1, and was seen by Dr. Durrant at 9.45 a.m., who then found one full bottle (twenty-five tablets) under his pillow, and one extra tablet, so that he had presumably taken 120 grains of veronal (also twenty grains of chloral and forty grains of Am. Brom.). He was then deeply comatose surface warm, respirations 32, pulse 102 per minute, mucus in throat, cornea insensitive, pupils moderately dilated, reflexes abolished ; at 12 noon was still comatose, pupils contracted, other conditions the same, but could be made to wince by slapping the face ; at 8 p.m. the same, unable to swallow ; 11 p.m. much the same, but pupils again dilated, and could swallow a teaspoonful at a time. At 9 a.m. on July 1st not quite so deeply comatose, pupils still dilated, could be made to wince by slapping the face, bowels not open, so ♏j of croton oil administered ; temperature 102·2°, pulse 110, reflexes still absent ; at 10.30 p.m. temperature 102·4°, finger nails dusky, breathing impeded by mucus, bowels not open, so calomel gr. v. administered. On July 2nd, at 9 a.m., better ; could be roused to speak a few words, pupils dilated, temperature 102·4°, pulse good, reflexes absent, bowels still not open, soon off to sleep again. Remained through the day in much the same condition, swallowed somewhat better, circulation better, and could be roused by speaking loudly. July 3rd, 9 a.m., condition much the same, temperature 102·8°, respiration 36, cough troublesome, bowels still not open, so ♏j of croton oil repeated ; cornea and pupils insensitive, not any more rousable, total muscular flaccidity, and still no reflexes ; at 2 p.m. not quite so well, respiration 36, pulse 120, temperature 102·8° ; at 8 p.m. temperature 103·2°, pulse 130, respiration 40, more comatose. On July 4th, at 2.30 p.m., temperature 104·2°, respiration over 40, pulse 150, died at 6.30 a.m. comatose.

Post-mortem examination : nothing found except intense congestion of lungs, liver, spleen, and brain, due, no doubt, to the asphyxial form of death. Mucous membrane of alimentary tract congested, otherwise normal.

A woman took 20 grains of veronal to relieve a headache. After sleeping for a couple of hours she awoke feeling dazed and stupid, quite unable to stand or walk, and with distinct double vision. During the evening the symptoms were more marked ; she was reeling and tottering, scarcely able to put one foot before the other, even with the assistance of her friends. At 10 p.m. she was in a semi-paralysed state, with sluggish pupils, diplopia, thready pulse, scanty urine, and normal temperature. Next morning she was drowsy and complaining of tingling sensations in the arms ; pulse 84, soft and compressible ; temperature, normal ; pupils, sluggish, though of about normal size ; reflexes, superficial and deep, quite gone. There was complete anæsthesia over the entire right lower limb, and on the left complete as far as the knee, and partial over the rest of the limb. There was also some anæsthesia over the trunk, but sensation in the arms was normal. The next day considerable improvement had taken place, sensation was normal over the trunk, left lower limb, and the upper portion of right lower limb. During the next few days the improvement continued and the reflexes became normal.[1]

A remarkable case is reported (*B.M.J.*, March 18th, 1933). A woman was taken ill on February 8th with " nerves," insomnia and colic. She was given 5 grains of medinal and paraldehyde. On February 10th, she took 6 ounces of sal volatile. Another doctor who was called in prescribed paraldehyde and 4½ grains nembutal. On the following night (11th) as she was still sleepless she was given 7½ grains medinal and on the 12th 6 grains nembutal.

On the morning of February 13th she awoke in a fresh bright condition ; shortly after she became restless and the nurse gave her 7½ grains medinal. She lapsed into unconsciousness and died at 3.30 in the afternoon, about eighteen hours after taking the nembutal and a few hours after the medinal.

Nembutal was found in the liver and kidneys, and the analyst attributed death to that drug or to the combined effect of the various drugs. Dr. Mapother of the Maudsley Hospital, gave evidence that he had given nembutal in over 6,000 individual doses without dangerous effects ; three doses, of 4½ grains each, in twenty-four hours was the usual practice, but 6-grain doses were given occasionally. He

[1] *B.M.J.*, February 2nd, 1907.

considered that nembutal was excreted within five or six hours, and that its effect was transient. The long period after giving the nembutal and the period of complete freedom from narcotic effects strongly suggests that nembutal was not the cause of death, yet the analyst found only nembutal with at most a trace of medinal on analysis.

Alexander[1] records a case of veronal poisoning in which 125 grains were taken. The patient was admitted to hospital in a state of unconsciousness in which he remained for four days. The face was flushed, the breathing stertorous, there was no normal reflex, the pupils were insensitive and strongly contracted. There was incontinence of urine and faeces. The patient regained consciousness on the fifth day, and thereafter made a complete recovery.

A pregnant girl attempted suicide by taking 3·84 grammes of allonal.[2] After twenty-four hours in a comatose state, fever and albuminuria developed and epileptic fits. After washing out the stomach, blood-letting and infusions of NaCl, cure followed in three days without abortion.

Poisoning by Acetanilide or Antifebrin ($C_6H_5.NH.CO.CH_3$)

Source and Method of Occurrence. This drug is a direct aniline derivative, and is given in doses of two to five grains but is no longer an official preparation. It is antipyretic in its action. It is by no means free from risk, though actual death from its use is not common. Hitherto those that have occurred have been either accidental or due to carelessness. Toxic symptoms have frequently followed its administration even in medicinal doses.

Toxicity and Fatal Dose. Aniline seems to hand on to all its derivatives the power which it itself possesses to destroy the red corpuscles of the blood and decompose the hæmoglobin, with the production of methæmoglobin which can be detected spectroscopically in the blood and which appears in the urine. Many of these drugs are oxidised in the body to para-aminophenol, and it is this substance which causes the formation of methæmoglobin. Snapper,[3] on the other hand, states that sulphæmoglobin is formed, and is responsible for the peculiar mauve colour of the cyanosed areas.

Young and Wilson[4] assert that the action of acetanilide is essentially the same as that of aniline. It forms para-aminophenol in the blood and urine, and it is this substance which gives the colour to the urine, and which probably accounts for the cyanosis observed. They consider that methæmoglobin is found neither in aniline, nor in acetanilide poisoning.

Judged by reported cases, the lethal dose may be somewhere between five and eleven grains. Fatalities have been reported with doses from 0·6–50 grammes.[5]

Symptoms. Large doses of acetanilide may cause collapse with shallow breathing, weak irregular pulse, cyanosis, cold sweat and circulatory failure. A man aged thirty-seven years was given sixty grains of acetanilide in six powders for headache, and he took them all within a few hours. When seen by his medical attendant (after what interval of time is not stated) he was slightly delirious and complained of pain in the head and in the left umbilical region. There were pyrexia, rapid heart,

[1] *B.M.J.*, July 5th, 1915.
[2] Remond, *Ann. de Méd. lég.*, 1925, Nr. 6, pp. 338–341.
[3] *Deut. med. Woch.*, 1925, p. 648.
[4] *Jour. Pharm. Exp. Therap.*, Baltimore, March 1926.
[5] Hanzlek, P. J., *J. Amer. Dental Assoc.*, 27 : 1505 (1940).

marked constipation, slight jaundice, nausea, and vomiting. Calomel in small doses followed by salts produced a copious but bloody motion and the urine was dark red. On the following day he was admitted to hospital. The pulse was seventy-eight, soft, and compressible; the temperature was 100·2° F.; the lips and nails were extremely cyanotic; and there was slight jaundice. He complained of pain in the left side of the abdomen, and there was tenderness in the epigastrium and in the region of the left kidney. The skin was moist and the gums were bluish. The urine was strongly alkaline and deep red—nearly black. The colour was shown to be due to hæmatoporphyrin. There was a small sediment which contained granular casts. On boiling, a large coagulum formed. On the day after admission only 150 cubic centimetres of urine were passed; after this there was complete suppression. There was great thirst, which was quenched with milk, but soon everything that was given was rejected and rectal feeding had to be adopted. Though nothing was given by the mouth for days the vomiting continued. Cough and expectoration were persistent. There was slight delirium, and the reaction of the pupils to light became more and more sluggish; in the end the pupils were widely dilated. The reflexes were first exaggerated, then they gradually disappeared. The extremities were constantly cold. The temperature fell slowly to normal on the fourth day and was subsequently subnormal, reaching 95·5° in the rectum on the evening before death, which took place on the eighth day of the illness. There was alternately constipation and diarrhœa, and forty-eight hours before death and twenty-seven hours after the suppression of urine blood-colouring matter and broken-down blood cells were constantly in the fæces, which previously were blood-stained only occasionally. The heart began to fail on the fourth day. The skin became more and more jaundiced. On the fifth day after admission mucous casts were passed. Examination of the blood showed destruction of the red corpuscles, which finally were reduced to 1,166,000 per cubic millimetre, while the leucocytes were 66,450 and the nucleated red cells 22,150. The alkalinity of the blood was diminished by 80 per cent. These blood changes are easily explained on the above view of the action of the drug.

Treatment. Empty the stomach and treat on general principles: artificial respiration and the administration of oxygen may be of use.

Post-mortem Appearances. At the necropsy on the above case acute nephritis and intestinal catarrh were found, but nothing to account for the melæna.[1] This nephritis is, however, not at all suggestive of poisoning by antifebrin, to which no characteristic appearances can yet be ascribed.

Analysis. Antifebrin may be extracted from acid aqueous solution (*e.g.*, in the Stas-Otto process) by ether or chloroform. Its melting point is 114° C.

(1) Sulpho-vanadic acid produces a brownish-red colour, which changes to dirty green. (2) If a drop of a solution of potassium dichromate is mixed with a drop of strong sulphuric acid on a colour-slab, and a fragment of antifebrin added, a red colour which changes to brown, and then to dirty green is produced. (3) Boiled with an aqueous solution of

[1] *Lancet*, 1, 243 (1902).

potash, antifebrin is decomposed into aniline and potassium acetate, which may be respectively recognised by appropriate tests. (4) Antifebrin may be distinguished from antipyrin by the absence of reaction on the addition of ferric chloride.

(5) An aqueous solution of antifebrin treated with bromine water gives a yellowish-white precipitate insoluble in dilute caustic potash, thus distinguishing it from a somewhat similar precipitate obtained with bromine water from aqueous solutions containing phenol or salicylic acid.

(6) If a little antifebrin is boiled with 5 c.c. of alcoholic KOH solution, and, after cooling, the mixture is warmed with a few drops of chloroform, phenyl isonitrile is produced and can be recognised by its intense offensive smell.

(7) *Indophenol Test.* Boil a little of the suspected material with a few cubic centimetres of concentrated HCl, evaporate to about $\frac{1}{2}$ c.c., cool, add 4 c.c. of 10 per cent. aqueous phenol solution, transfer to a test-tube, and add a few drops of freshly prepared bleaching powder solution (calcium hypochlorite). In presence of acetanilide, a reddish-violet colour is produced which deepens on standing and turns to a deep indigo-blue on addition of ammonia. If the ammonia is added carefully, to form a separate layer, the blue colour is confined to the ammoniacal layer. Note that calcium hypochlorite, phenol and ammonia will themselves give a blue colour, but not the violet-red prior to addition of the ammonia.

Acetanilide is excreted in the urine partly as such and partly as *p*-amino-phenol both free and conjugated with glycuronic or sulphuric acid. This derivative may be identified by the method given on p. 585.

Cases. The first fatal case occurred in 1896.[1]

The victim, *æt.* 22, took a " Daisy powder " at 1 p.m. Seen in less than five minutes, and then said that the headache for which she had taken the powder was worse, and said she felt as though she would go out of her mind. Screamed, went into a sort of fit. Was conscious at intervals when not convulsed. At 1.30 the face was very cyanosed and the patient appeared to be dying. Artificial respiration brought back colour and improved respiration, slowing it and making it less laboured. Pulse very rapid, thready, and uncountable. Gave rational answers. Intense pain in legs. No convulsions from 1.30 to 1.45, when she was much better. She died about 2 p.m., *i.e.*, in one hour. Convulsions unlike those of strychnine.

Dr. Chestnutt said there was no doubt in his mind that death was due to poison by the " Daisy powder." The public analyst for the East Riding confirmed generally the doctor's evidence. He had purchased twenty-one powders and weighed them ; the weights varied from 4·60 to 10·89 grains and they consisted of pure acetanilide.

On January 25th, 1900, an inquest was held at Liverpool on a little girl, *æt.* 6 years 10 months, who died from taking a " Daisy powder." The directions were that she was to have half a powder, but by mistake took a whole one.

Dr. Baxter said he found the child breathing with great difficulty, with lips and skin very blue in appearance. He had no doubt the child died from paralysis of the centre of respiration, caused by taking too much of the powder.

Estéve[2] records the following case :—

After taking 0·5 gram of acetanilide a day for about a month, the patient became comatose, with pale face, cold extremities and cyanosed lips. Acetanilide passes into the urine as para-aminophenol sulphate, and in this case was present eight days after the patient had stopped taking the drug. The accumulation of the drug was probably due, in part, to some failure of the excretory organs. It was

[1] *Pharm. Jour.*, 1896, 2, p. 14.
[2] *Bull. Soc. Pharm.*, Bordeaux, 1923, No. 4 ; *Ann. Chim. Anal.*, 1 4, 6, 272 274.

conclusively shown that the cyanosis was not due to the presence of methæmoglobin and hæmatin in the blood, or the general poisoning to the presence of aniline as an impurity in the acetanilide.

Poisoning by Exalgine, or Methylacetanilide

Source and Method of Occurrence. This is another coal tar derivative used as an analgesic and sedative ; it is not official ; its dose is stated to be half to three grains, and consequently it is dangerous for self-medication. In the following case[1] the patient took 150 grains and survived :—

A Chinese male adult was taken to the Government Civil Hospital, Hong-Kong, on July 31st, at 11 a.m. ; he stated that he had taken some medicine out of a tin purchased in the town, and this tin was produced. It was the usual one-ounce tin stamped with the words " Exalgine " and " Merck." The patient was quite unconscious, intensely livid, with pin-point pupils and a full, bounding pulse. His temperature was 100·8° F. He had vomited once. He was given thirty grains of salicylic acid by the nasal tube and one-fiftieth of a grain of atropine hypodermically, and was put to bed. He remained in much the same condition, so two hours later he was given one-hundredth of a grain of atropine, with small quantities of milk and strong coffee. The urine was examined and contained albumen. During the night he again had one-hundredth of a grain of atropine. Next morning he was out of danger though his colour and pupils were not quite normal, but the urine was now free from albumen. He was somewhat weak for several days, and his temperature varied between normal and 100·8°, though this might have been due to the primary syphilis from which he was found to be suffering. He apparently purchased and took the medicine to open his bowels, though it was found impossible to ascertain why and where he bought it.

In the following case five grains caused severe symptoms :—

A single woman, aged thirty, extremely thin, was under my care for severe asthma and consequent insomnia. On May 3rd of this year she was given by a friend, without my knowledge, five grains of exalgine. Within five minutes " she screamed out, becoming perfectly stiff." Twenty minutes later, when I saw her, she was profoundly unconscious, her respiration being very shallow and infrequent and rapidly failing. The lips and finger-tips were markedly cyanosed and the extremities were cold ; the pupils were widely dilated and fixed ; the knee-jerks were absent ; the pulse was ninety-five, small and feeble. One-fifth of a grain of apomorphine was given at once, but failed to produce emesis. Free stimulation with brandy and coffee, vigorous flagellation, faradisation, and occasionally artificial respiration were employed for three hours, at the end of which time the cyanosis had nearly disappeared and respiration was fairly well established. The patient, when roused, was incoherent and failed to recognise her surroundings, rapidly relapsing into unconsciousness. About an hour afterwards there was an evident tendency to heart failure, the pulse dropping repeatedly below fifty and becoming irregular and feeble. Ether was given at regular intervals hypodermically. Nine hours after taking the drug the pulse and respiration were both good, the pupils reacted well, and the knee-jerks were present. No urine could be drawn off with a catheter. The next day the patient was perfectly well except for aphonia, which lasted for some hours. The points of interest in the case appear to be the rapidity of the onset of the toxic effects and the evident danger in giving even moderate doses of exalgine—a respiratory poison—to asthmatics.

Poisoning by Phenacetin (Acet-phenetidin : p-ethoxy-acetanilide)

Source and Method of Occurrence. This drug is a coal tar derivative with antipyretic and analgesic qualities ; its official dose is five to ten grains ; it is said to be free from unpleasant effects, but in one case three doses of eight grains each caused very alarming symptoms.

Reid[2] records a case of phenacetin poisoning in which the drug had been taken for five months. The patient had blueness of the lips, tongue,

[1] *Lancet*, 1899, 2, p. 890.
[2] *Jour. Amer. Med. Assoc.*, 1926.

ears and finger-tips. She complained of weakness and fainting and pains in the head. Respirations 70 to 80, and shallow. Voice whispering. Pulse small, systolic pressure 110, diastolic 65. An electrocardiagram revealed normal rhythm and no evidence of heart-block.

J. H., aged 40, on the morning of July 23rd, 1895, complained of neuralgic headache. He was ordered powders, which were subsequently ascertained to be phenacetin eight grains, every three hours. He took the third dose about 5 p.m., and shortly afterwards, while at his tea, he began to feel very ill ; his wife noticed that his face was very pale. He was taken upstairs with difficulty and put to bed. When seen soon afterwards he was complaining of shivering, inspiratory dyspnœa, and profuse sweating from the forehead. The face was of a dark, almost mahogany colour, somewhat swollen ; on the back of each hand was a wheal of similar colour, pyriform in shape, with the apex at the styloid process of the radius, and the base extending from the metacarpo-phalangeal joint of the thumb to that of the third finger. The shirt was thrown open at the neck, disclosing another wheal of much lighter colour on the right shoulder, about the size and shape of the palm of the hand. There were no wheals anywhere but on these exposed portions of the body. At intervals the patient had sudden jerking of the whole body. He was extremely anxious, and afraid that he was about to die.

The mercury would not rise in a thermometer graduated down to 95° ; pulse very feeble, 100, regular. There was a systolic, mitral murmur, and sibilant and sonorous rhonchi all over the chest. The urine appeared on inspection to be normal.

The points of interest in the case were the distribution of the rash only on portions of the body exposed to the air, the extreme depression of the temperature, and the profuse perspiration from the forehead, with dryness of the rest of the skin. He was well next day.[1]

For another case, where seventy-five grains were not fatal, *vide* *B.M.J.*, 1904, 1, 545.

In one case a man took for headaches on several occasions, during fourteen days, a dose of 1 gramme. One night after such a dose he was seized with vomiting and collapse and died next morning. The autopsy showed methæmoglobinæmia and brown coloration of all the viscera.

Analysis. Phenacetin is a white crystalline powder slightly soluble in cold water (1–150), more soluble in hot water, alcohol (1–16). It melts at 135° C.

Phenacetin is extracted by ether or chloroform from the final aqueous acid solution in the Stas-Otto process. In the indophenol test, performed as described under antifebrin, it gives first a carmine-red colour, changed to blue by ammonia.

The boiled solution of phenacetin in concentrated hydrochloric acid, cooled, filtered and treated with a drop of chromic acid solution, gives a red colour. It is important to note that *p*-amino-phenol gives no colour with this test which therefore serves to distinguish them.

It is excreted in the urine as **para-aminophenol,** which can be detected as follows :—

Acidify the urine with HCl, boil to hydrolyse conjugated compounds with glycuronic and sulphuric acids, and decolorise with animal charcoal ; To a few cubic centimetres add five drops of 3 per cent. chromic acid : a brownish colour is obtained. Another portion warmed and treated with a few drops of ferric chloride solution gives a brownish-red colour. Para-aminophenol in the urine is, however, not proof of the ingestion of phenacetin, since acetanilide and many other aromatic amino compounds cause a similar reaction in the urine.

[1] *B.M.J.*, 1896, 1, p. 146.

Poisoning by Antipyrin (Phenazonum ; phenyl-dimethyl-pyrazolone)

Source and Method of Occurrence. This is another coal-tar derivative introduced into medicine for its power of reducing fever. It is a very powerful drug and dangerous for self-administration. Numbers of cases of unpleasant and even dangerous symptoms arising from its use have been recorded, and several deaths. It has not yet been used for homicidal purposes.

The drug is official as phenazonum, its dose being given as five to ten grains.

Iodo-, bromo-, ferri-, and sali-pyrin are non-official combinations of it ; hypnal (chloral hydrate antipyrin), pyramidon (dimethyl amino antipyrin), and tussol (antipyrin mandelate) are also trade names for certain of its compounds.

Symptoms. These vary materially in individual cases, but extreme collapse, coldness, and depression of the heart's action are common features. Methæmoglobinæmia is said not to occur. The following supplies a typical example[1] :—

A man, aged twenty-four, who was suffering from " neuralgia," applied to a chemist for ten grains of antipyrin. This was taken in the shop at the time. Within a quarter of an hour after taking the dose the patient felt very ill. Shortly afterwards his face was cyanosed, his lips and nose swollen and blue and his eyes almost closed from swelling of the eyelids. His skin was cold and clammy ; he was sweating and his pulse was 128, very weak, small, and compressible. The pupils were widely dilated. He was very much alarmed, and expressed himself as being in fear of impending death. He vomited a little mucus mixed with saliva. He walked to a consulting-room, distant nearly a mile from where the dose was taken, and was given five grains of carbonate of ammonia, one-fiftieth of a grain of digitalin, one-fiftieth of a grain of strychnine, and half an ounce of vinum aurantii. After resting for a quarter of an hour his condition improved so far as the symptoms of cardiac depression were concerned. The pulse grew fuller and steadier, the feeling of faintness passed off, and he expressed himself as feeling better. He was still perspiring freely, and the pupils were moderately dilated.

Vide also *B.M.J.*, Epit., 1899, 2, p. 7.

Analysis. Antipyrin may be extracted by chloroform from both acid and alkaline solution, but it is preferable to make alkaline before shaking out, and the substance is usually obtained in the alkaloid fraction in the Stas-Otto process. It may be separated from any alkaloid present by evaporating the solvent and dissolving in hot water. The melting point is 111–113° C.

Tests. With ferric chloride a dark-red colour is produced, which is destroyed by mineral acids in excess. When antipyrin is heated with a solution of bleaching-powder a brick-red precipitate is formed. If a little potassium nitrite is dissolved in water, and excess of strong sulphuric acid is added, the nitrous acid set free produces a green colour with antipyrin : a colour is given by all pyrazolones, but pyramidone gives a blue. The urine from patients taking antipyrin yields the ferric chloride reaction on simple addition of the reagent. Antipyrin is precipitated by most of the alkaloidal group agents.

Cases. For a case of severe rash, etc., following antipyrin, *vide Lancet*, 1897, 1, p. 309.

REFERENCE

Gourin, " Des Accidents dus a l'Antipyrin." Paris, 1893.

[1] *B.M.J.*, 1896, 1, p. 269.

Poisoning by Aspirin (Acetyl-Salicylic Acid) and Salicylates

Aspirin is very extensively used as an anti-pyretic and analgesic, and in the treatment of various rheumatic conditions. Ordinarily, it can be taken with complete safety, and very excessive dosage or a marked degree of personal idiosyncrasy are required before it can be considered "poisonous."

Such idiosyncrasy does occur, however, and occasional cases are met with in which small doses of aspirin, well within the average therapeutic range, produce alarming symptoms. These allergic symptoms include angio-neurotic œdema, urticaria, œdema of the mucous membranes with hypersecretion, vomiting, excessive salivation, bronchial spasm, cyanosis, weak intermittent pulse, and coldness of the extremities. Death has occurred in five minutes from œdema of the glottis. True aspirin poisoning, however, results only from really massive dosage, and several hundred grains are usually required to produce a serious or fatal effect.

The main symptoms of poisoning by aspirin or salicylates are largely an indication of the state of acidosis and ketosis which is produced. There may be a latent period of several hours during which a mild pyrexia may develop. The victim feels faint and nauseated and may vomit. There may be profuse perspiration. Vertigo, ringing in the ears, deafness and impaired vision are commonly experienced, and headache may be severe. The breathing is deep and rapid, and acetone may be smelt in the breath. The urine is strongly acid, contains acetone, albumen, and frequently a trace of bile resulting from a mild nephritis and hepatitis. There is great thirst. The pulse rate is accelerated, the force of the beat is impaired, and the rhythm may become irregular. Delirium, progressive weakness and collapse may lead eventually to death in severe cases.

Sodium salicylate is slightly less toxic than aspirin, but the effects of overdosage are similar.

Methyl salicylate poisoning is less common, and presents a somewhat different clinical picture, in which gastro-intestinal irritation and convulsions figure more prominently.

Toxic Dose. There are approximately twenty fatal cases of suicidal aspirin poisoning in England and Wales each year, and to those can be added an even larger number of unsuccessful or half-hearted attempts. The incidence of aspirin poisoning is therefore quite considerable, but the toxic or lethal dose of aspirin is extremely difficult to estimate. In a good proportion of the fatal cases, the quantity consumed has been exactly 500 grains, *i.e.*, the entire contents of the commonly dispensed bottle containing a hundred 5-grain tablets. Deaths have resulted from the consumption of this quantity[1] and, since considerable numbers of unabsorbed and unaltered tablets have been recovered from the stomach at the post-mortem examination of such cases, it is apparent that much less than 500 grains *can* prove fatal : for example, Neale[2] records a fatal case after 200 grains. On the other hand, there are many cases recorded of recovery after the ingestion of 400, 500, 600, and 750[3] grains of aspirin, even in the absence of active treatment. These figures refer, of course, to dosages which will endanger the life of an adult. Less serious toxic

[1] Biddle, E., *B.M.J.*, 1 : 1365 (1938).
[2] Neale, A. V., *B.M.J.*, 1 : 109 (1936).
[3] Charters, A. D., *B.M.J.*, 1 : 10 (1944).

manifestations will result from much smaller doses of either aspirin or sodium salicylate ; and proportionately smaller doses are dangerous for children. In one case, a child aged $2\frac{1}{2}$ years was given 10 grains of aspirin, four hourly, to a total amount of 150 grains. Severe symptoms occurred, including convulsions, and the child died in spite of treatment. The skin of the entire body was covered with petechial hæmorrhages, which were found also under the scalp and in the various organs of the body. Microscopic examination showed widespread hæmorrhages throughout the organs, and a marked toxic hepatitis.[1]

Stiel[2] has reported a case of chronic poisoning after the daily ingestion of 20 grains of aspirin for seven years. Oedema of the tongue and fauces was present, along with conjunctivitis, diarrhœa, vomiting, and widespread urticaria.

The fatal dose of methyl salicylate (oil of wintergreen) is about 1 ounce. Less than half an ounce may cause the death of a child. In a recorded case of fatal poisoning of a child aged $2\frac{3}{12}$ years by one ounce of methyl salicylate, hæmorrhages were found throughout the viscera at post-mortem examination.

Treatment. The stomach should be thoroughly emptied by emesis and repeated lavage with warm water and 5 per cent. sodium bicarbonate solution. The acidosis should be counteracted by the administration of alkaline fluids by mouth and intravenously. The patient must be kept warm and at rest. Stimulants may be necessary, and the aspiration of cerebro-spinal fluid by lumbar puncture has been recommended.

In the case which Charters reported the ketosis was treated by the injection of insulin 40 units combined with oral administration of 40 grammes glucose. Oral doses of 20 grammes glucose was given every two hours for 12 hours, then insulin 10 units morning and evening before meals followed by 40 grammes glucose three hours later. This patient who had ingested exactly 750 grains of aspirin made an uninterrupted recovery.

Post-mortem Appearances. These are by no means characteristic. there is usually evidence of irritation of the gastric mucosa and sometimes petechial hæmorrhages into the mucus and sensuous membranes. The kidneys usually show signs of cloudy swelling of the tubules and hyperæmia and these are signs of hepatitis.

Analysis. Tissues, etc., may be acidified slightly and extracted with water. The filtered extract containing the aspirin is evaporated to dryness and the residue extracted with ether in which aspirin is soluble. The residue from evaporation of the ethereal extract will therefore contain the aspirin. Aspirin crystallises in needles. It gives no colour with ferric chloride, but is hydrolysed by boiling with sodium hydroxide solution to sodium acetate and sodium salicylate. If this mixture is acidified slightly with acetic acid, the salicylic acid gives a violet colour with ferric chloride. After acidification, too, the salicylic acid can be extracted with ether. The residue from evaporation of this extract, heated with a little methyl alcohol, and a few drops of concentrated sulphuric acid gives methyl salicylate (oil of wintergreen) recognisable by its smell.

[1] Troll and Menton, *Amer. Jour. Dis. Child*, 69 : 27, 1945.
[2] *Practitioner*, 99 : 293, 1917.

Since aspirin is partly (and often considerably) hydrolysed in the body, urine should be tested for salicylic acid as well as for unchanged aspirin. Sometimes hydrolysis occurs even in the intestine, so the same procedure should be followed in testing intestinal contents, or vomited matter. The salicylic acid may be separated by steam distillation after acidification of the material.

Group 6. ARTIFICIAL SUBSTANCES NOT IN GROUP 5

This group is one purely of convenience in description, though in it several smaller groups occur between the members of which there is real affinity and likeness.

Sub-Group 1. Hydrocyanic acid and the cyanides.

Sub-Group 2. Organic acids acting as irritants in large doses. Acetic, tartaric and picric acids.

Sub-Group 3. Substances containing a nitrite group, all acting as vaso-dilators, nitroglycerine ; amyl nitrite, cordite, and roburite.

Sub-Group 4. Substances, principally of coal tar origin, with an antiseptic action (for carbolic acid *vide* " Corrosives ").

The following are noticed :—

1. Coal-Napththa.
2. Benzene.
3. Naphthaline.
4. Naphthol Camphor.
5. Iodoform.
6. Resorcin.
7. Formaldehyde.

Sub-Group 5. A number of substances the bond of connection between which is their close relationship to aniline.

1. Aniline.
2. Nitrobenzene.
3. Phenyl hydroxylamine.

Sub-Group 6. Paraffin, Pyridine, Piperazine.

Sub-Group 1.—Poisoning by Hydrocyanic Acid (HCN) and the Cyanides

Source and Mode of Occurrence. At one time most of the commercial cyanogen compounds were by-products obtained in the purification of coal-gas. Crude coal-gas contains 25–100 grains of "cyanogen" (chiefly as hydrocyanic acid and ammonium cyanide) per 100 cubic feet. This is removed, usually as sodium (or, less often, potassium) ferrocyanide, which, until recently, was the source of all other manufactured cyanides and other cyanogen derivatives. Potassium cyanide is still prepared from this source, but sodium and calcium cyanides are now of much more frequent occurrence and of much greater industrial importance. Sodium cyanide is prepared by the action of gaseous ammonia on sodium in presence of carbon ; calcium cyanide by fusing sodium chloride with calcium cyanamide. (This substance is obtained by the action of ammonia on calcium carbide : it is not a cyanide, but is toxic.) Hydrocyanic acid itself is prepared by the action of sulphuric acid on any metallic cyanide.

Hydrocyanic Acid (Prussic Acid) is a liquid, miscible with water in all proportions. It is used medicinally in 2 per cent. solution (official dose 2 to 5 minims of the solution). It is used in various chemical processes, but its main commercial application is in fumigation, for it is extremely poisonous to all forms of animal life. It is not germicidal in moderate

concentrations. Curiously enough, it stimulates the growth of many plants. Hydrocyanic acid is decomposed by water, especially rapidly in neutral or alkaline solution, with liberation of ammonia. This necessitates special precautions during transport, since the liberation of gas may cause bursting of the containers.

The fumes of hydrocyanic acid or cyanogen are frequently used to fumigate rooms to free them from bugs, lice, etc., and to fumigate ships to rid them of vermin and for general purposes of a similar nature, and agriculture and horticulture.

A great many cases of accidental poisoning from these procedures have been recorded.[1] The failure to take suitable precautions in a firm of fumigators were fined £300.[2]

Potassium Cyanide contains 43 per cent. of the cyanide radicle. The commercial salt is usually about 90 per cent. pure, and contains chloride, carbonate, cyanate, etc., of potassium. It is used in a few chemical processes, in electro-plating, and in photography, but for most purposes has been superseded by the cheaper sodium and calcium cyanides. Like all soluble cyanides, it usually smells of hydrocyanic acid, since it is partly decomposed by water, giving the free acid. Commercial potassium cyanide has a local chemical action on the skin, and if the skin is broken may cause serious effects. Accidents of this kind have occurred in the practice of photography and of electro-plating.

Sodium Cyanide is usually about 96 per cent. pure. It is extensively used in metallurgy, in electro-plating, for case-hardening steel, in tanning, in the manufacture of dyes, etc., and as a source of hydrocyanic acid in fumigation. Opportunities for accidental poisoning are thus present in all these processes.

Calcium Cyanide, commercial, is about 50 per cent. pure. It is used chiefly in the mining industry and as a source of hydrocyanic acid.

Silver Cyanide (used in silver plating) is insoluble in water, but is readily decomposed by the acid of the gastric juice, liberating hydrocyanic acid.

Organic Cyanides. A number of plants contain considerable amounts of cyanogenetic glucosides, and traces of hydrocyanic acid have been detected in a great many plants (*e.g.*, by Rosenthaler, *Biochem. Z.*, 1922, 134, 215, in 56 of 88 plants). Amygdalin (a glucoside which is hydrolysed to hydrocyanic acid, benzaldehyde and glucose by acids or by the enzyme emulsion which occurs along with it) is found in bitter almonds, in the kernels of the peach, plum, apricot and cherry ; in the seeds of the apple and pear ; in the cherry laurel plant ; and in other plants of the genera Prunus, Cerasus, Sorbus, etc. Hydrocyanic acid is thus present in cherry laurel water (0·17 per cent.), essential oil of bitter almonds (2 to 4 per cent., or even more) ; essence of peach and cherry kernels, etc., all of which are used for giving flavour or odour to confectionery and cakes. "Almond flavour," containing one drachm of essence of peach kernels

[1] McNally, W. D., *Medical Jurisprudence and Toxicology*, 1939, 266.
[2] *Lancet*, 1 : 891 (1939).

to seven drachms of rectified spirit, is reported to have caused thirty-one deaths within four years in England and Wales. Poisoning from this source, however, is now rare, as the crude oil is a scheduled poison and its sale is restricted.

Other glucosides producing HCN are found in the Java bean, the lotus, millet and linseed. It is stated to be found in a free state in the juice of the cassava. Sudan grass contains derivatives of hydrocyanic acid, and has caused poisoning of cattle in South Africa.

Toxicity and Fatal Dose. Hydrocyanic acid and the cyanides must be considered as highly poisonous substances. Hydrocyanic acid is a general protoplasmic poison, and kills by its paralysing action on the central nervous system, and also by the direct paralysis of the automatic power of the heart. In non-lethal doses there is an intial stimulation of the medullary centres, but this is of short duration. Hydrocyanic acid inhibits tissue respiration, preventing the action of cytochrome, a pigment concerned in the transference and utilisation of oxygen. In cyanide poisoning, therefore, the tissues are unable to take up the oxygen of the blood, which remains bright red. The venous blood, in fact, may contain nearly as much oxygen as arterial blood. It is possible, though unproved, that this prevention of oxidation may be the sole toxic action of hydrocyanic acid. The rapidity of its action would, in that case, be attributed to the stoppage of oxidative processes in vital centres. It prevents ferment action, and inhibits the action of blood on hydrogen peroxide.

Lehmann has proved that the minimum lethal dose lies at about 0·05 per thousand of HCN in the atmosphere breathed. One to five mgrm. per kilo of the body weight is fatal to animals.

Hydrocyanic acid was used by the French in shells under the name of Vincennite during the 1914-18 war, but its use was discontinued, as it was impossible to obtain a lethal concentration under field conditions.

The minimum fatal dose of the anhydrous acid may be placed at about one grain or approximately fifty minims of the official dilute acid. We recently investigated a case in which three young adults received a measured dose of hydrocyanic acid due to an error in dispensing. The dose calculated from an analysis of the mixture was equivalent to 1·04 grains of HCN. One of the persons died, the other two recovered. In the case in which death occurred the young woman walked away from the dispensing counter without difficulty to a room about 150 yards away, where she was found unconscious five minutes later. She was moaning with froth at the mouth and gurgling respiration. She became completely comatose within twenty minutes and died within thirty minutes of taking the dose. The other two cases recovered. The fatal case indicates clearly that quite definite movements and purposive acts may be performed after a fatal dose. In another case[1] a stout, healthy man swallowed a dose, nine-tenths of a grain, by mistake, and remained insensible for *four hours*, when he vomited and began to recover. The vomited matters had *no odour* of the poison, showing that, if not concealed by other odours, the whole of the acid must have been absorbed. Banks published a case in which a female recovered after swallowing thirty drops of prussic acid.[2] Little reliance can be placed upon most records of doses for these

[1] *Med. Gaz.*, vol. 36, p. 104.
[2] *Edin. Med. Surg. Jour.*, vol. 48, p. 44.

reasons: (*a*) the acid is very volatile and (*b*) very easily decomposed by exposure to light for a length of time, and hence when a person takes a dose of the ordinary liquid preparations of the acid the actual amount of hydrocyanic acid ingested is very problematical.

From the facts hitherto observed, we shall not be far wrong in assuming that *about fifty minims* of pharmacopœial acid (2 per cent., *i.e.*, *one grain of anhydrous acid*), or an equivalent portion of another preparation, would commonly suffice to destroy the life of an adult. This is the nearest approach that we can make to the *smallest fatal dose*. In *R.* v. *Bull*[1] a question arose respecting the minimum fatal dose of this poison.

The accused, a young medical man, was charged with the manslaughter of his mother, a woman, *æt.* 66. He had prescribed for her prussic acid to relieve sickness. He procured a bottle of Scheele's acid, said to contain one drachm. He administered four minims to the deceased in the morning, and it appeared to benefit her. In the evening he gave to her another dose, amounting, according to his statement, to "seven drops." The deceased went upstairs, became insensible, and died in a few minutes. When the bottle was examined twenty-five minims remained in it; hence thirty-five minims were alleged to be missing, but the druggist who sold the acid poured out the quantity conjecturally, and the bottle was found to have a broken cork. The strength of the acid had not been determined. In these circumstances the prisoner was acquitted.

In this case the Court desired to know the relation of drops to minims, but no satisfactory answer could be given. The size of a drop materially depends on the nature of the liquid, the mouth of the bottle, and the rapidity of the measurement. Seven drops of Scheele's acid dropped from a small phial measured seven minims. There can be no doubt in the above case that the poison caused death, and unless we assume that seven drops or minims will destroy life, which is not probable, the deceased must have taken a much larger dose than the accused had intended. Scheele's acid is twice the strength of the pharmacopœia acid: the latter contains 2 per cent. of prussic acid, and its official dose is two to six minims.

Of KCN five grains have caused death in a quarter of an hour, but recovery has followed the swallowing of forty grains. The toxicity of the salts of hydrocyanic acid seems to depend, partly, upon the rapidity with which they liberate hydrocyanic acid in the stomach—*i.e.*, upon the acidity of the gastric juice and upon the nature and amount of the stomach contents.

Duration. Hydrocyanic acid is one of the most rapidly fatal poisons known to chemists. It has caused death within three or four minutes. A dose of five grains of KCN, equal to two grains of prussic acid, has proved fatal within three minutes. One of the strongest diagnostic points in poisoning by this acid or the cyanides is the rapidity with which the symptoms appear: they commonly appear more rapidly than with any other solid or liquid poison, and are only equalled in this respect by those of CO and a few other gaseous bodies. In some instances there may be loss of consciousness within a *few seconds*; in others, certain acts indicative of volition and locomotion may be performed, although requiring for their performance several *minutes*. When the dose is two drachms and upwards

[1] *Lewes Aut. Ass.*, 1860.

we may take the average period for death at from **two to ten minutes.** In Hicks' case twenty grains of Scheele's acid destroyed life within twenty minutes.

But although death does not commonly ensue until after the lapse of a few minutes, sensibility, and consequently a power to perform certain acts of volition and locomotion, may cease in a few seconds. The time at which this loss of muscular power is supposed to take place has frequently become an important medico-legal question ; and on the answer to it the hypothesis of suicide or murder in a particular case may rest. The reason for the difference is not clear, but the following is a case in point[1] :—

A maidservant was found dead in bed. The body lay at full length, with the head turned a little on one side, the arms crossed over the trunk, and the bedclothes pulled up smoothly to the chin. Under the clothes, on her right side, lay a corked phial which was wrapped in paper and contained three and a half drachms of hydrocyanic acid. From the size of the bottle it was probable that four and a half drachms of the poison had been swallowed, and the question arose whether the girl after taking this quantity of the poison could have corked the bottle, wrapped it up, and adjusted the clothes.

A man was suspected of having murdered her, but as several cases were quoted at the trial showing that in other instances of undoubted suicide similar acts had been performed the prisoner was acquitted. Such cases afford considerable difficulty and no general lines can be laid down for their elucidation ; each case must be considered in detail as it occurs.

The following cases emphasise the fact that movement is possible :—

On November 4th I received an urgent message to see Miss A. A few minutes afterwards I found the patient, a girl, aged twenty, at the foot of the staircase, breathing stertorously, unconscious, convulsed, with widely dilated pupils, no pulse at the wrist, and cold. Her mother informed me that Miss A. had gone upstairs to change her dress, intending at the same time to do some photographic work. She shortly afterwards heard her rush out of her room calling "Mother!" immediately afterwards falling downstairs, convulsed and very sick. There was a strong odour of bitter almonds in her breath and vomit, and I came to the conclusion she had in some way poisoned herself with cyanide of potassium or prussic acid. She was perfectly well half an hour before the occurrence.

I removed her to bed and at once injected $\frac{1}{100}$ gr. of digitalin and $\frac{1}{50}$ gr. of strychnine, followed by two syringefuls of brandy, and also commenced artificial respiration and general friction. Shortly afterwards there was some feeble pulsation at the wrists, and with the aid of a battery (which had by that time been obtained), fresh injections of brandy, hot bottles, and mustard over the heart, the patient gradually rallied, not, however, before she had several alarming relapses. She was several times sick and slightly convulsed. The pupils were widely dilated the whole time. She did not recover consciousness until fully three hours after the accident, but then rapidly rallied, and in the evening only felt faint and sick.

On searching her bedroom a large piece of cyanide of potassium, and a penknife with which she had been scraping it to a powder, were found. How she managed to convey a portion of this powder to her mouth she is quite unable to say, as she remembers nothing of the whole occurrence, although there must have been a momentary period of consciousness after taking the poison, as she was able to rush out and cry for help.[2]

A woman, who at the time was under medical treatment, took by mistake a teaspoonful of a solution of cyanide of potassium, containing about seven grains of the salt. Immediately after taking it she complained of a severe burning pain in the stomach, and feeling as if the bowels were about to act. She went to the water-closet, and her strength left her. She was removed to bed, and speedily became unconscious. It was found impossible to introduce anything into the stomach. She died in less than an hour. There was no convulsion before death, but a sudden convulsive action of the body took place after the heart had ceased

[1] *Lancet*, 1903, 1.
[2] Leonard C. Dobson, *B.M.J.*, 1896, 1, p. 17.

to beat. The appearance of the body was so natural even on the day following death, that some of her friends supposed that there might still be life.[1]

A woman swallowed an ounce and a half of a solution of cyanide of potassium, used for photographic purposes. The quantity taken amounted to five grains. In two minutes she became unconscious, the whole of the body was slightly convulsed, and the pupils of the eyes were dilated. She foamed at the mouth, the pulse was small and feeble, and there was spasmodic closure of the jaws. Nevertheless, as she had lost some teeth, there was sufficient space for the introduction of the tube of the stomach-pump, within five minutes after she had taken the poison. She died within twenty minutes.

Symptoms. In some cases, in animals at any rate, no symptoms are to be observed, death supervening with startling rapidity. When a patient is seen within a minute or two he may be perfectly insensible, the eyes fixed and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the skin cold and covered with a clammy perspiration: there is convulsive breathing at long intervals, and the patient appears dead in the intermediate period; the pulse is imperceptible, and involuntary evacuations are occasionally passed. The respiration is slow, deep, gasping, and sometimes heaving or sobbing.

When the dose is large, the breath commonly exhales a strong odour of the acid, and this is also perceptible in the room. Convulsions of the limbs and trunk, with spasmodic closure of the jaws, are usually met with among the symptoms; the finger-nails have been found of a livid colour, and the hands firmly clenched. The breathing is generally convulsive, but when the coma or insensibility is profound, it is sometimes stertorous. The breathing in prussic acid poisoning is of a special and peculiar character. The intervals between the respirations are prolonged, followed by tedious and spasmodic respirations, the inspirations being short, and the expirations very protracted.

A medical student took a drachm and a half of Scheele's prussic acid. He was heard to call out once or twice and to fall from the sofa to the floor, and when picked up was found to be already insensible. Hilton Fagge saw him a little later—fifteen to thirty minutes after the poison was taken. He was then lying on a sofa, quite insensible. The limbs were paralysed and lay in any position in which they were placed: and they were free from all rigidity. The jaws were clenched. The pupils were normal. There were no convulsions, nor had there been any. The face was not livid. The pulse was very rapid, the respirations very infrequent. Cold water dashed on the face produced at each application deep respiratory efforts. An attempt to administer an emetic failed, in consequence of inability to swallow. Strong ammonia applied to the nostrils failed to produce the slightest stimulation. After a time the breathing became slower—seven in a minute; and it was distinctly stertorous. The pulse became slower, and markedly feebler; and now cold affusion failed to produce any effect. The respirations fell to four in a minute, the face became blue, and the pulse imperceptible at the wrist. Breathing ceased in from an hour to an hour and a half after the poison was swallowed. There was a marked odour of prussic acid in the room; but it was not noticed at first whether the breath smelt of the acid.[2]

When a small dose (*i.e.*, about thirty minims of a weak acid) has been taken, the individual has first experienced weight and pain in the head, with confusion of intellect, giddiness, nausea, a quick pulse, and loss of muscular power: these symptoms are, however, sometimes slow in appearing.

Vomiting has been occasionally observed, but it is more common to find foaming or frothing at the mouth, with suffusion or a bloated

[1] *Boston Med. and Surg. Jour.*, December 11th, 1856, and *Brit. and For. Med. Rev.*, 1857, vol. 19, p. 498.

[2] Guy's Hosp. Rep., 1869, p. 259.

appearance of the face, and prominence of the eyes. If death results, this may be preceded by tetanic spasms, opisthotonos, and involuntary evacuations.

The potassium salt has a bitter taste, producing first a sense of coldness on the tongue followed by a feeling of constriction, and burning heat in the throat.

The symptoms which the cyanides produce are similar to those occasioned by prussic acid : insensibility, spasmodic breathing, convulsions, and tetanic stiffness of the jaws and body. They appear in a few seconds or minutes, and run through their course with great rapidity. An acid state of the stomach, by liberating prussic acid from the salt, is thought to hasten its effects.

When the gas is inhaled in lethal concentrations there is a sense of constriction about the throat, giddiness, confusion, throbbing in the head, palpitation and laboured respiration. Consciousness is rapidly lost, and if the victim is not at once removed from the vapour, he dies from respiratory failure.

Chronic poisoning may be produced by the continued inhalation, over long periods, of very low concentrations of hydrocyanic acid vapour. The symptoms are those of the early stages of fatal poisoning, though in milder degree. Small amounts of hydrocyanic acid can apparently be decomposed in the animal body—probably to the non-toxic thiocyanic acid.

Treatment.—The action of the poison is very rapid and therefore no time must be lost in commencing treatment. In general, rapid evacuation of the stomach by lavage should be aimed at, using some substance which will destroy any remaining poison. Permanganate of potash (5 grains to the pint) or 5 per cent. solution of sodium thiosulphate or hydrogen peroxide may be used for this purpose. Absorbed poison is difficult to neutralise, but an attempt may be made to combat its effect by the use of methylene blue intravenously as described hereunder. Stimulants such as coramine or cardiazol and digitalis may be used, and artificial respiration, with oxygen and carbon dioxide inhalation, may be of value in combating the failure of circulation and respiration.

Hanzlik and Richardson[1] have discussed the value of various antidotes such as sodium nitrite, sodium thiosulphate and methylene blue. Although sodium nitrite and thiosulphate solution are of more value than methylene blue in animal experiments, they have other features which suggest that methylene blue is the antidote of choice in human cases. Methæmoglobin combines firmly with cyanide to form cyanhæmoglobin which is non-toxic, therefore any drug which causes the formation of methæmoglobin will act as an antidote to cyanide. The danger arises from the fact that methæmoglobin does not act as an oxygen carrier and if a considerable proportion of hæmoglobin is so altered the patient may die of asphyxia. Therefore, though sodium nitrite is theoretically a valuable antidote, it is too dangerous to warrant its use owing to the excessive amount of methæmoglobin formed and the rapid fall in blood-pressure which it causes.

They recommend the immediate intravenous injection of 50 c.c. of 1 per cent. solution of methylene blue containing 1·8 per cent. sodium sulphate, repeated if necessary until a total of 200 c.c. has been used.

[1] Hanzlik, P. J., and Richardson, A. P., *J. A.M. A.*, 102, 1740 (1934).

In cases of poisoning by inhalation, immediate removal to the open air and artificial respiration offer the best chance of saving life.

Trautman[1] considers that methylene blue in inhalation cases is of no avail if a lethal or near lethal dose has been inhaled.

Some experiments with hydrocyanic acid vapour have been made by Violle.[2] A concentration of 2 gr. of HCN per cubic metre of air will kill an animal in a few minutes. It is stated that animals which have previously had a dose of glucose (by injection or by mouth) can breathe this toxic atmosphere for more than an hour without ill effects. The glucose apparently enables them to neutralise hydrocyanic acid, and the tissues are able to deal with relatively enormous quantities. These findings, if confirmed, should have a practical application; since glucose might be used as a prophylactic by those whose work brings them into contact with the gas itself—*e.g.*, in cleaning ships of rats—and those who have to handle cyanide compounds.

It is of interest in connection with Dr. Violle's experiments to recall that an attempt is alleged to have been made on Rasputin's life on December 16th, 1916, by putting HCN in some wine three hours before he drank it. No effect was produced, and experiments made indicated that the sugar in the wine inhibited the action of HCN to a certain extent.[3]

Antal[4] has investigated the action of cobaltum nitricum oxydulatum in this respect. When cyanides are present in the stomach of an animal, and a solution of this cobalt salt is introduced, a harmless potassium cobalticyanide (K_3CoCy_6) is formed. That the cobalt salt can render absorbed cyanides harmless is due to the rapidity of its absorption and to the small quantity needed to make large quantities of cyanide inert.

Antal records a series of experiments to show the antidotal properties of the above-named cobalt salt. When rabbits or dogs are given a lethal dose of cyanide, and $\frac{1}{2}$ to 1 per cent. solution of the cobalt salt is introduced into the stomach, no poisonous effects are produced. Experiments are also related which show that this solution, subcutaneously injected, will also neutralise the effects of absorbed cyanide. He concludes that in man, to render inert already absorbed cyanide, $\frac{1}{2}$ per cent. solution of the above-named salt (10 to 30 c.c.) should be injected subcutaneously: at the same time the same solution should be given by the mouth or, if that is impossible, passed into the stomach by means of a tube to neutralise any cyanide still present there.[5]

Post-mortem Appearances. The body when seen soon after death often exhales the odour of prussic acid; but if it has remained exposed before it is seen, and if it has been exposed to the open air or in a shower of rain, the odour may not be perceptible; again, the odour may be concealed by tobacco-smoke, peppermint, or other powerful odours. In a case in which a person poisoned himself with two ounces of the acid, and his body was examined twenty-eight hours after death, the vapour of prussic acid, which escaped on opening the stomach, was so powerful that the inspectors were seized with dizziness. In cases of suicide or accident, the vessel out of which the poison has been taken will commonly

[1] Trautman, J. A., Public Health Reports 48, 1443 (1933); *J. A.M. A.*, 102, 217, 1934.
[2] *Lancet*, July 10th, 1926.
[3] *Presse Médical*, Paris, October 9th, 1926.
[4] *Physiol. Studien aus d. Univ. Budapest*, 1895.
[5] *B.M.J.*, Epit., 1895, 1, p. 44.

be found near; but there is nothing to preclude the possibility of a person throwing it from him, or even concealing it if the symptoms should be delayed. Owing to the great volatility of the poison, the vessel, if left uncorked, may not retain the odour when found.

Externally, the skin often has a lifelike colour but is sometimes livid, or violet coloured; the nails are blue, the fingers clenched, and the toes contracted; the jaws firmly closed; foam or froth forms about the mouth, and the eyes have been observed to be wide open, fixed, glassy, very prominent and glistening, and the pupils dilated; but a similar condition of the eyes has been observed in other kinds of violent death.

Internally, the venous system is gorged with liquid blood; in many cases the blood in the veins is bright red and arterial in character, and when the muscles are incised they have a bright red colour such as is seen in carbon monoxide poisoning, the smell of cyanide may be noticed in the muscles. The larynx, trachea and bronchi usually contain fine froth and the lungs are congested and smell of hydrocyanic acid. The stomach and intestines may be in their natural state; but in several instances they have been found more or less congested. The mucous membrane of the stomach is often reddened throughout, and may present the appearance met with in cases of arsenical poisoning. Areas of punctate hæmorrhages may be observed. The brain is usually congested and a faint odour of hydrocyanic acid may be noticed.

In one case of poisoning by bitter almond oil, nine hours after death no odour was perceptible in the chest, head, heart, nor in the venous blood with which the system was gorged. The lungs and heart were healthy. The vessels of the brain were congested, and there was a general effusion of serum on the hemispheres. The lining membrane of the stomach was much congested. On opening it the bitter-almond odour was quite perceptible. In the case of a boy who died in a quarter of an hour, on inspection there was a pallor of the face, with lividity of the depending parts; the lungs were congested; the odour of the poison was perceptible only in the abdomen, and very distinctly in the contents of the stomach. The mucous coat of the stomach was generally pale, but there were some patches of ecchymosis scattered over it. The essential oil and prussic acid were detected in it. In a case which proved fatal in three hours the skin was partially livid, the blood fluid, and the membranes of the brain as well as the lungs were gorged. The contents of the stomach had a strong smell of the oil, and the mucous coat towards the intestinal opening had a red appearance. The other organs were healthy.[1]

Analysis. Prussic acid is limpid, like water; its solutions are acid to litmus, and its vapour has a peculiar odour which is not always perceptible at first, even from concentrated solutions of the acid. [Inhalation of the vapour rapidly produces giddiness, insensibility, and other alarming symptoms.] Solutions of cyanides (the common inorganic cyanides are white solids) are usually alkaline to litmus, and have the same odour as solutions of the free acid. Potassium cyanide is deliquescent.

Preliminary Treatment. Hydrocyanic acid or cyanides may be *detected* in stomach contents and other organic liquids or in viscera without distillation. For *quantitative estimation*, however, it is necessary to

[1] *Lancet*, 1845, July 12th, p. 40.

separate the hydrocyanic acid by distillation. The cyanogenetic glucosides (amygdalin, etc.) liberate HCN on being warmed with acids. They are therefore detected in the following procedures. They are differentiated from HCN itself or from simple cyanides by odour, etc.

To detect cyanide, the fluid or minced tissue is simply placed in a wide-mouthed bottle closed by a watch-glass and standing in warm water. The bottle should be filled to within about an inch of the mouth. The material to be examined, if not already acid, should be acidified with tartaric acid (to ensure liberation of free HCN from its salts)—mineral acids are to be avoided since they may produce hydrocyanic acid by decomposition of the thiocyanates which are present in saliva. In presence of sulphuretted hydrogen—*i.e.*, in putrefied material—it is desirable also to add lead carbonate. The underside of the watch glass is wetted with one or other of the reagents used in testing for hydrocyanic acid (see below), so as to absorb the hydrocyanic acid vapour. A number of successive tests may be performed on the same sample.

In using the distillation method, the sample is again acidified slightly with tartaric acid, and the flask, connected to a condenser and receiver, is heated in a boiling brine bath until sufficient of the fluid has distilled over. For quantitative work it is advisable to distil half the contents of the flask, but to detect hydrocyanic acid it is sufficient to collect about a quarter of this amount. The receiver may merely be cooled in water or ice, or may contain potassium hydroxide or silver nitrate solution to fix the hydrocyanic acid.

It is desirable to ascertain that the relatively non-poisonous ferro- and ferricyanides are absent, since they may be decomposed during the distillation, and yield free hydrocyanic acid. The acidified material should therefore be tested with ferrous sulphate and ferric chloride, which give deep blue colours with ferrocyanide and ferrocyanide respectively.

Mercuric cyanide is not decomposed by tartaric acid. When its presence is suspected it is necessary to distil with hydrochloric acid.

Autenrieth (*Arch. Pharm.*, 1893, 231, 99) recommends distilling the material with a considerable amount of sodium hydrogen carbonate (instead of tartaric acid) and adding hydrogen sulphide solution as well if mercuric cyanide is suspected.

Tests. 1. *The Silver Test.* Silver nitrate, added to a solution of hydrocyanic acid or of a cyanide, gives a dense white precipitate which rapidly flocculates and settles. The precipitate is characterised by the following reactions :

(*a*) It is insoluble in dilute nitric acid, but dissolves in boiling concentrated nitric acid (*i.e.*, after pouring off the clear supernatant fluid and boiling the drained precipitate with concentrated nitric acid).

(*b*) It evolves hydrocyanic acid when digested with hydrochloric acid.

(*c*) It does not darken on exposure to light.

(*d*) When *dried* thoroughly and heated in a small reduction-tube it forms cyanogen, which may be burned at the mouth of the tube and recognised by its rose-red flame with a blue halo.

The test is capable of detecting ·004 mg. of hydrocyanic acid in 10 c.c. of solution. The watch-glass method gives a rough idea of the amount of hydrocyanic acid present ; moderate amounts give an immediate opaque white film ; smaller amounts give a slowly produced film starting at the edges of the nitrate solution and spreading, in a few minutes, over the whole surface ; very small amounts, reaching the silver nitrate slowly

and greatly diluted with air, produce microscopic crystals (visible under high power magnification) consisting of slender prisms with oblique ends. [This test may be used for detecting the vapour of hydrocyanic acid in air.]

2. *The Prussian Blue Test.* To 2 c.c. of the liquid to be tested add 2 or 3 drops of a ferrous sulphate solution, and a similar amount of sodium or potassium hydroxide solution. Warm the mixture (in which a dirty green or brownish precipitate has appeared) to about 50° C. for a few minutes, acidify, and, if no blue colour appears, add a drop of ferric chloride solution (this is only necessary in the unlikely event of the ferrous sulphate solution being free from ferric salt). In the presence of hydrocyanic acid, the liquid becomes dark green or blue, or a precipitate of Prussian Blue may appear, according to the amount of cyanide present. It is said (Anderson, *Z. Anal. Chem.*, 1916, 55, 459) that a coloration is given by ·04 mg. of HCN in 10 c.c. of solution.

In the watch-glass method the glass is moistened with caustic soda or potash solution, which absorbs hydrocyanic acid, and the test is carried out after a few minutes' exposure.

Silver cyanide, obtained in a previous test with silver nitrate, or by absorbing the distillate in silver nitrate solution, may be decomposed by hydrochloric acid, and the liberated hydrocyanic acid submitted to the Prussian Blue test after absorption of the vapour in alkali.

3. *The Thiocyanate Test.* A solution of sodium or potassium sulphide, or of yellow ammonium sulphide, is added drop by drop to the test solution until the yellow colour persists. The mixture is evaporated to dryness on a water bath (it becomes colourless on warming), preferably after addition of a few drops of caustic soda solution. The residue, in the presence of hydrocyanic acid, contains thiocyanate. It is dissolved in a little water, and acidified with dilute hydrochloric acid. A red colour produced by addition of a drop of ferric chloride solution then indicates the presence of hydrocyanic acid in the original test solution. The reaction is claimed to be ten times as delicate as the Prussian Blue test—*i.e.*, to detect ·004 mg. of HCN in 10 c.c. of solution. It may be made even more delicate by shaking the final solution with ether, which extracts the red substance (ferric thiocyanate) and becomes red itself. The colour is discharged by mercuric chloride.

In the watch-glass method the hydrocyanic acid is absorbed by yellow ammonium sulphide on the watch glass, and the remainder of the procedure carried out as described.

The test is of great value, performed in this way, or for detecting hydrocyanic acid in air, on account of its great delicacy. It is capable of detecting a concentration of one part of hydrocyanic acid vapour in 400,000 parts of air.

4. *Colorimetric Tests.* Hydrocyanic acid gives an orange-yellow colour with dry sodium picrate, a greenish-blue colour with guaiacum and copper sulphate solution, and a pink colour with phenolphthalein and copper sulphate in alkaline solution. The colours may be obtained by exposing test-papers impregnated with the various reagents. The tests have been put on a quantitative basis by Katz and Longfellow (*J. Ind. Hygiene*, 1923, 5, 97) and used for the detection and estimation of hydrocyanic

acid in air. They easily detect the acid in a concentration of 25 parts per million. The paper by these authors must be consulted for details of the process and of the preparation of reagents. (See also Allen's "Commercial Organic Analysis," 5th ed., Vol. VIII (1930, p. 495).

5. *Spectroscopic Test.* Oxhæmoglobin in dilute solution (*e.g.*, blood diluted with about twenty times its volume of water) is oxidised by a little potassium ferricyanide to methæmoglobin. Half of this solution is kept as a control and a drop or two of the test solution (*e.g.*, distillate obtained in the preliminary procedure outlined above) is added to the other half. The presence of HCN is indicated by the immediate appearance of a bright red colour readily recognised in comparison with the dark brown of the control tube. Spectroscopic examination of the tube containing HCN shows a broad absorption band between D and E. The reaction is very delicate and is claimed to give positive results with hydrocyanic acid in a concentration of ·003 mg. per 10 c.c. of solution.

6. *Oil of Bitter Almonds* (or peach-nut oil) is colourless when pure, but is usually yellow. This colour, with the smell of bitter almonds, renders it liable to be confused, on mere inspection, with *nitrobenzene* (oil of mirbane). Oil of bitter almonds has a hot burning taste and a feebly acid reaction (again like most samples of nitrobenzene). However, on standing for some time, and more rapidly on warming with acid, oil of bitter almonds liberates hydrocyanic acid which responds to the usual tests. Also, when mixed with a few drops of concentrated sulphuric acid, the oil assumes a rich crimson colour, which is changed to yellow on dilution with a large amount of water. The impure oil is rapidly oxidised to benzoic acid, the pure oil only very slowly. The pure oil has a sp. gr. of 1·043. It is slightly soluble in water (about 3 per cent.) and easily soluble in alcohol and ether.

7. *Estimation of Hydrocyanic Acid.* Hydrocyanic acid may be estimated by titration of the distillate mentioned above with a solution of silver nitrate (·01 N $AgNO_3$ is usually convenient). The distillate in this case should be well cooled during collection, and should contain a few drops of potassium hydroxide solution.

A useful method for the quantitative estimation of hydrocyanic acid has recently been published by Aldridge.[1] In the absence of thiocyanates (which react similarly) it can be made on urine without preliminary treatment or on blood plasma after deproteinisation by trichloracetic acid. Since thiocyanic acid is not volatile, it is safest to separate hydrocyanic acid by distillation, using the distillate for the following procedure. To 1 ml. of solution (containing up to 3 mg. of hydrocyanic acid) acidified with acetic or trichloracetic acid, 0·5 ml. of saturated bromine water is added. This, almost instantaneously, produces cyanogen bromide. Excess of bromine is removed by addition of 0·5 ml. of 1·5 per cent. sodium arsenite. To the solution are then added 5 ml. of pyridine reagent (25 ml. pure redistilled pyridine and 2 ml. concentrated hydrochloric acid diluted to 100 c.c. with distilled water) and 0·2 ml. of 2 per cent. benzidine solution. An orange colour appears at once and changes rapidly to red. After 10 minutes at room temperature it is compared with the colour of a standard solution of hydrocyanic acid similarly treated (or with a calibration curve previously prepared from standards).

Duration of Hydrocyanic Acid in the Body. Lwow (*Ann. d'Hyg.*, 1882, p. 571) has detected cyanide in the body 100 days after death. Zillner (*Viertljahrschift f. gericht. Med.*, 1882, 35, 193) after four months. Autenrieth (*Ber. Pharm. Ges.*, 1910, 20, 432) detected it in an exhumed body forty-three days after burial. In quantitative experiments, in which bitter almond water or potassium cyanide was added to a putrefying mixture of blood, stomach and intestines, he was able to recover 41·4–63·3 per cent. of the added cyanide after sixty days.

Case. The following case of recovery after swallowing at least twenty grains of potassium cyanide is reported by T. R. Wiglesworth.[1] A miner was seen to drink something from a bottle, throw the bottle away, and fall down in convulsions, foaming at the mouth.

I saw him not less than fifteen minutes after drinking the fluid. He was lying insensible on his back on the floor (where he had been placed). His face was greyish-blue in colour, the mouth covered with foam, and the jaws tightly clenched, so much so that I broke one of his teeth in forcing open his mouth to insert the stomach-tube. The eyes were intensely injected and fixed, and the pupils widely dilated, but the conjunctival reflexes were not entirely absent. The breathing was pectoral, the inspirations were jerky and sounded similar to a faint hiccough. The pulse was small and rapid. The arms were slightly flexed at the elbow, the fingers tightly contracted, and the thumbs pressed firmly into the palms; the abdominal muscles were rigid. There was no escape of urine nor of fæces.

As soon as I was able to separate his teeth sufficiently, I introduced the stomach-tube, and washed out his stomach with clean water, and then injected a mixture of sulphate of iron, carbonate of potash, and pure ether, which was retained for about five minutes, when he vomited, the vomit being distinctly stained blue. I then poured a stream of cold water over the back of the neck and spine, and over the region of the heart. His pulse and breathing rapidly improved, but the muscular contractions remained until 6.30 p.m., nor did he regain full consciousness until 8.15 p.m., when he spoke and answered questions quite rationally, although he denied all recollection of taking the poison. I then left him, giving him a mixture of sulphate of iron and carbonate of ammonium to take every hour. He complained next day of his teeth feeling tender, but was otherwise well.

Two cases of recovery from large doses of potassium cyanide are reported by W. F. Stevenson.[2] In 1862 a man swallowed the greater part of a solution containing an ounce of the commercial cyanide which he had dissolved for the purpose. Taafe applied the stomach-pump, and cold affusion freely. In two hours the man vomited, and from that time rapidly recovered.

In 1876 a patient was admitted into Guy's Hospital suffering from the effects of a handful of bitter almonds, which he had eaten. The symptoms were those of prussic acid poisoning, from which he recovered.

Mr. McCarthy related to the editor a curious case that occurred some years ago, in which a gentleman was fatally poisoned in Ireland by drinking the first glassful out of a new bottle of noyeau liqueur. There had accumulated in the bottle and floated to the top a sufficient quantity of prussic acid to kill.

In one case a woman swallowed about seventeen drops of the essential oil of bitter almonds, and she died in half an hour. The first symptoms observed in this case were strong convulsions, the deceased throwing her arms about as if in pain. A boy, *æt.* 13, swallowed a quantity of the oil; he was found lying on the floor motionless and insensible; the face pale, the eyes opened and fixed, the pupils dilated, and he was rolling about and panting for breath; the pulse at the wrist was imperceptible; he died in a quarter of an hour without any convulsions appearing. A man, *æt.* 20, swallowed about two ounces of the oil. A person present saw him fall suddenly while in the act of swallowing: he made a loud cry, gave one deep expiration, and died.

[1] *B.M.J.*, 1897, 1, p. 1039.
[2] *Lancet*, 1871, 1, p. 806.

In another case a woman, *æt.* 46, who had been in the habit of using the almond essence for flavouring confectionery, swallowed about half an ounce (equal to thirty drops of the oil). She died in less than half an hour.

Poisoning by Ferro- and Ferri-cyanides

Potassium ferrocyanide (yellow prussiate of potash) has generally been regarded as non-toxic, but cases of severe poisoning or death have followed its ingestion.[1]

Ipsen investigated a case in which an old man poisoned his wife with repeated doses of ferrocyanide of potassium. The symptoms came in paroxysms coincident with the poison administered and recovery took place very rapidly. In one attack, however, she died : the salt was easily found in her organs by the Prussian blue test.[2] [The blue colour is produced on simple addition of ferric chloride solution.]

Poisoning by Thiocyanates (Sulphocyanides)

Potassium thiocyanate has been fairly extensively tried in hypertension and good effects have been claimed after several days but the drug often causes toxic symptoms similar to those of iodism. There is often intense fatigue, cramps, vertigo, nausea and vomiting. If continued a toxic psychosis may develop.

The drug is not converted into cyanide in the body. It is excreted by the kidney unchanged, but very slowly. Death has been reported in several cases.[3] [4]

It is a normal constituent of saliva in minute quantities.

The following suicidal case is recorded :—

On Wednesday, February 28th, the borough coroner (Mr. H. Saunders French) held an inquest at the Hoop Hotel, Bridge Street, Cambridge, on the body of Florence Eliza Stearn, aged thirty-five, who had died after taking sulphocyanide of ammonium. Mr. David John Rygate, surgeon, deposed to being called in to see the deceased at 9.30 p.m. on Sunday, February 25th, when he was informed that, about 2 p.m., she had taken sulphocyanide of ammonium. The father of the deceased then told witness the story of the events of the day. Witness ordered the deceased to bed, and at 2.30 a.m., on Monday, he found her unconscious, with rigidity of the muscles of the arms and jaws. By injections of ether attempts were made to revive her, but convulsions ensued and the deceased expired on Monday evening at 6.30. A *post-mortem* of the body had been made. He was of opinion that the deceased died from convulsions, whether caused by taking poison or not he was not absolutely certain, as he was not sufficiently competent to detect the small amount of poison which had been taken. The remedies applied got rid of the greater part of the compound, and he had not much doubt that the deceased died of the after-effects of the small quantity which got into the system. The jury returned a verdict of "Death from convulsions, probably caused by taking a quantity of sulphocyanide of ammonium whilst of unsound mind."[5]

Analysis. Sulphocyanides (=thiocyanates) give a red colour, extractable by ether, on addition of ferric chloride solution. They are decomposed by sulphuric acid to hydrocyanic acid.

[1] Volz, *Vrtljschr. f. ger. Med.*, 1877, 26, 57 ; Schlichte, *Med. Corr. Bl. d. Württemb. ärztl. Verein*, 1895, 65, 25.
[2] "Gerichtsärztliche und Polizeiärztliche Technik," Wiesb., 1914, p. 716.
[3] Garvin, C. F., *J. A.M. A.*, 112 : 1125 (1939).
[4] Goldring, W., *New York Med. Jour.*, 31 : 1322 (1931).
[5] *Pharm. Jour.*, March 17th, 1894, p. 791.

Sub-Group 2.—Poisoning by Acetic Acid

Toxicity. This acid is by no means a dangerous drug, but it has been used for suicidal purposes, and many cases of accidental death have come under our notice from drinking concentrated acetic acid by mistake. Common *vinegar*, which contains only 4 or 5 per cent. of acetic acid, has often been taken in large doses without injurious consequences. The concentrated acid is highly corrosive and causes acute irritation. In one case, a female, aged nineteen, was found dying on the highway. She suffered from convulsions, complained of pain in the stomach, and died in a short time. On inspection, the stomach was found neither softened nor corroded, but its mucous membrane near the pylorus was almost black. The mucous glands were prominent, and the vessels were filled with dark coagulated blood.

Analysis. The material to be examined, *e.g.*, gastric contents or vomitus, is acidified and distilled in a current of steam. The distillate, containing acetic acid, is then exactly neutralised with NaOH and evaporated to dryness. The residue, containing any acetic acid as the sodium salt, may be submitted to the following tests. [*Vinegar*, which may be regarded as an organic mixture containing a small proportion of acetic acid, may be examined in the same way.] (1) A portion of the residue heated with sulphuric acid gives the odour of acetic acid. (2) An aqueous solution gives a red colour with ferric chloride, destroyed by HCl or H_2SO_4. (3) The residue, boiled with alcohol and a few drops of concentrated H_2SO_4, yields ethyl acetate, recognised by its odour. (4) A little of the residue, heated with arsenious acid in a dry, hard glass tube, gives cacodyl, recognisable by its disagreeable smell.

Vinegar, as it exists in commerce, may contain a small quantity of sulphuric acid, and occasionally traces of lead and copper. In general it is easily recognised by its odour.

Case. In the following case it might be presumed that the acetic acid was mainly responsible, as red oxide of mercury is not a very active poison, at any rate as a corrosive. On the other hand, a mixture of the two would result in the formation of mercuric acetate, a *soluble* mercuric salt, which no doubt played an important part in determining the fatal issue.

C. S., a well-nourished girl, aged seventeen, was admitted to the Hereford Infirmary at midday on November 22nd, having swallowed half an hour previously one ounce of acetic acid, to which she had added an unknown amount of the red oxide of mercury. Evidence showed that the bottle she brought to the chemist's for the acid was perfectly clean. Afterwards there was found on the bottom of the broken bottle a red deposit, which on analysis proved to be red oxide of mercury. The inference drawn was that in addition to the ounce of acetic acid, thirty grains at least of the oxide had been taken, as it was proved that one drachm of acetic acid dissolved five grains of the oxide.

The symptoms were stertorous breathing, vomiting of frothy fluid, no blood; pain in throat and stomach, and frequent bloody evacuations from the bowel, many copious clots being passed. The patient was conscious all through, but very collapsed: there were no convulsions and very slight charring of the mouth. Treatment consisted of magnesia suspended in emulsion, lime water, and olive oil, with hypodermic injections of ether. The patient died, apparently from haemorrhage, at 4.30 p.m. next day, seventeen hours after taking the poison.

On *post-mortem* examination eight hours after death the œsophagus was inflamed and congested, and the stomach intensely so. At the pyloric end was a large patch, quite black and charred, with the mucous membrane quite destroyed; the organ was empty. The duodenum was also inflamed in patches, but not ulcer-

ated. Both ventricles of the heart were contracted and empty. The trachea and larynx were inflamed as far down as the bifurcation of the bronchi. There was no peritonitis.[1]

Several fatal cases have occurred through the incautious use of the acid for domestic purposes.[2]

Poisoning by Tartaric Acid

Symptoms and Appearances. Tartaric acid is not commonly regarded as a poison; but at least one case has occurred in which there was no doubt that it acted as an irritant and destroyed life. The case referred to was the subject of a trial for manslaughter.[3] The accused gave the deceased, a man, aged twenty-four, *one ounce* of tartaric acid instead of aperient salt. The deceased swallowed the whole, dissolved in half a pint of warm water; he immediately exclaimed that he was poisoned; he complained of having a burning sensation in his throat and stomach as though he had drunk oil of vitriol, and stated that he could compare it to nothing but being all on fire. Soda and magnesia were administered with diluent drinks. Vomiting set in and continued until death, which took place nine days afterwards. On inspection, nearly the whole of the alimentary canal was found highly inflamed. The accused admitted that he had made a mistake, and tartaric acid was found in the dregs of the cup. The jury acquitted the prisoner. Another case of poisoning by this acid, with a report of the results of analysis, has been published by Devergie.[4] This case gave rise to a controversy between Orfila and Devergie, the points in dispute relating to the processes for the detection of the acid in the stomach and tissues.[5]

The following fatal case has been reported[6] :—

A woman aged 67 owned to having taken "about two teaspoonfuls" of tartaric acid, but it is probable that a larger dose may have been taken. The remnant of tartaric acid taken from the room was on examination found to be free from admixture or impurities. No other irritant poison was discoverable in the house, nor did any of the circumstances in any way point to the likelihood of any drug other than tartaric acid being concerned.

The symptoms at the outset were acute abdominal pains, vomiting and diarrhœa. Pain and vomiting appear to have supervened almost immediately after the administration, and diarrhœa set in some few hours later.

On the fourth evening the patient became delirious and remained so until her death. About the fourth day also the pulse began to fail markedly. The temperature from this time forward was subnormal. On the day before death, diarrhœa, which had been checked, recommenced. The urine was twice tested during the illness, and found to be strongly acid but free from albumen or sugar.

A necropsy was made fourteen hours after death: *rigor mortis* was then strongly present. The serous surface of the small and large intestines was found coated with a layer of soft, butterlike lymph. The serous surface of the stomach appeared free from this. There were some patches of similar inflammatory product on portions of the peritoneum which lay in contact with the inflamed gut, and in addition there was a small similar patch between the base of the left lung and the diaphragm. The surfaces of the mesentery were free from lymph. There was no free fluid in the peritoneal cavity. In the œsophagus there were a few shallow erosions. In the stomach were found several patches of subserous hæmorrhage, and the mucous membrane was covered with a layer of rather tenacious mucus. The mucous surface of the whole intestine showed signs of recent inflammation, but there was nowhere any sign of chronic disorder. The transverse and descending

[1] *B.M.J.*, 1896, 1, p. 19.
[2] *Vide Lancet*, 1909, 1, p. 656; also *Lancet*, 1905, vol. 2, for additional fatal cases.
[3] *R.* v. *Watkins*, C. C. C., January, 1845.
[4] *Ann. d'Hyg.*, 1851, 2, 422.
[5] *Ann. d'Hyg.*, 1852, 1, 199, 382; and 2, 230.
[6] *B.M.J.*, 1 : 1321 (1923).

colon appeared perhaps to have suffered more severely than any other part of the gut. The lungs were emphysematous, the liver rather fatty, and the spleen soft. The heart, kidneys, pancreas, vermiform appendix, and pelvic organs were normal. There was no evidence anywhere of tuberculosis.

At the inquest it was stated in evidence by the chemist who sold the tartaric acid that the poor used tartaric acid very largely in concocting summer drinks, and he further expressed his opinion that a good many of them looked upon tartaric acid and cream of tartar as different names for the same article. The coroner's jury returned a verdict in this case of " Accidental poisoning from an overdose of tartaric acid."

Poisoning by Picric Acid or Trinitrophenol ($C_6H_2(NO_2)_3OH$)

Source and Method of Occurrence. Picric acid is an explosive obtained by the nitration of phenol. It was at one time largely used as a dressing for burns, and toxic effects have been produced, one case at least ending fatally.[1]

If taken by the mouth 15–30 grains causes acute toxic symptoms, acute gastro-enteritis, hæmorrhages, nephritis and toxic hepatitis. The skin and conjunctivæ is enlarged yellow mostly due to the drug but partly due to jaundice. The urine may be coloured port wine or yellowish green. Headache and vertigo with nausea and vomiting and skin rashes occur after absorption from the skin.

Symptoms. The following case[2] illustrates the effects of a toxic dose :— A girl, aged sixteen, endeavoured to commit suicide by swallowing about 300 grains of picric acid mixed with water. Violent pain in the stomach and repeated vomiting speedily occurred and diarrhœa soon followed ; the sclera and the skin were coloured an intensely dark yellow, almost brown : the pupils were moderately dilated and reacted feebly to light ; the fingers were spastically stretched and bent at the metacarpo-phalangeal articulations. The urine was ruby-red in colour ; it contained neither albumen nor bile pigment ; a slight sediment formed which partially consisted of brown-stained epithelium ; the stools were fluid and ruby-red in colour. Both urine and fæces contained picric acid in considerable amount ; six days after the ingestion of the poison traces of it were present in the urine. In a few days the discoloration of the skin diminished and the patient was quite well at the end of a week. In another case symptoms of poisoning occurred from the application of about six grains of powdered acid to the vagina ; in one hour the skin was discoloured and erythematous, and the urine was red ; pain in the stomach and the kidneys with somnolence were amongst the symptoms ; recovery took place, but the skin was discoloured for a week, and the erythema persisted for eleven days. A teaspoonful of picric acid has been swallowed without other ill effect than violent vomiting and purging.

The most frequent symptoms of chronic poisoning (in munition works, etc.) are yellowness of the skin and itching dermatitis. The swallowing of the dust may lead to abdominal pains, diarrhœa, loss of appetite and weight, and dizziness.

Treatment. The stomach should be evacuated and well washed out, and elimination promoted by diuretics and, if necessary, aperients : morphine will probably be required to relieve pain and cramps.

[1] Alexander, *Med. Press and Circular*, 1912, 112.
[2] *Wiener med. Wochenschr.*, 1880. (Dixon Mann.)

Analysis. Organic matter should be acidified with hydrochloric acid and digested with alcohol over a water-bath ; after filtration the alcoholic extract is evaporated to a syrup, taken up with boiling water, filtered, acidified with sulphuric acid, and shaken out with ether, chloroform, or amyl alcohol. Dragendorff directs attention to the fact that if chloroform or benzene are used for extraction, the solution, though containing picric acid, will be almost colourless ; if ether or amyl alcohol is used it acquires a yellow tint. The extract is evaporated to dryness and the residue dissolved in water and tested.

An aqueous solution of picric acid, gently warmed with a little concentrated solution of potassium cyanide, changes to a deep blood-red colour. Ammonia-copper sulphate yields a green precipitate with picric acid. Basic lead acetate gives a yellow precipitate. Allow a solution of picric acid to stand with a solution of stannous chloride in HCl, and then add a little ferric chloride solution ; a blue colour is produced. Warm (on the boiling water-bath) a solution of picric acid with an alkaline solution of glucose ; the mixture becomes orange-red in colour. A piece of white silk or wool allowed to remain a short time in a solution of picric acid is dyed yellow ; the colour is not discharged by subsequent washing in water. In similar circumstances cotton is not dyed.

Picric acid melts at 122·5° C. and can be sublimed without decomposition.

Cases. Many cases in which unpleasant effects have followed the use of picric acid as a dressing have been published.

A boy, aged nine years, was scalded on September 15th, 1903,[1] on the left chest and flank. The burn was of the first degree, with only one or two small blisters. An ointment composed of picric acid and vaseline (half a drachm to the ounce) was applied on lint. The scalds were dressed daily. On September 18th he was drowsy, there was a slight icteric tint of the conjunctivæ, and the face and palms were a little yellow. The pulse was 120, but the temperature was normal. On the 19th the drowsiness continued, there was vomiting, and the temperature was 101·2° F. On the 20th and 21st the temperature fell to 99° and there was mild diarrhœa. The whole of the skin was yellow, but especially that of the face, palms and soles. The hair along the border of the scalp was deep yellow. The urine contained albumen and was of the colour of dark port wine, giving the impression of hæmoglobinuria, but neither blood nor bile pigment was present. The burns were dressed with boric ointment. On the 22nd the vomiting and diarrhœa continued ; in the morning the temperature was 103° ; in the evening it fell to normal. On the 23rd the discoloration of the skin was less ; the burn was sodden-looking. To dry it up gradually a strip of lint two and a half inches by one inch covered with picric acid ointment was applied in the loin. On the 24th there were vomiting and headache and the temperature was 101·4°. There was a bright red blotchy general eruption which was here and there papular. The urine was again deeply coloured. The picric acid dressing was removed. On the next day the rash was fading. In a second case a man, aged forty-five years, scalded his shin and the same ointment was applied. In four days the skin and conjunctivæ were yellow and in six days the urine was like that in the previous case. There were slight diarrhœa and headache. The symptoms rapidly subsided when the ointment was discontinued. Toxic symptoms in these cases appear to have been due to using too strong a preparation of picric acid. It has been extensively used in 1 per cent. solution without untoward results.[2]

Another case is as follows :—

A man, aged thirty-five, took what he at the time thought to be some powdered sulphonal, a drug he was in the habit of administering to himself, but which proved

[1] *Scottish Medical and Surgical Journal*, December 1903.
[2] *Lancet*, 1904, 1, p. 247.

to be picric acid, much used in his trade as a dye. The amount swallowed was a pennyworth, about a tablespoonful. No immediate ill-effects followed; in fact, it was some hours before he found out his mistake.

The following morning he complained of slight frontal headache, pain over the abdomen and across the loins. He was deeply jaundiced. His urine contained bile and blood. The stools were natural.

Next day the jaundice had slightly diminished, though the abdominal and lumbar pain persisted. In addition there was lachrymation, injected conjunctivæ, profuse mucous discharge from the nares, and sore and irritable fauces. The urine still contained bile and blood.

Three days later he was practically well, though still slightly jaundiced. At no time were the stools paler than normal, but the urine for some days contained both bile and blood.[1]

A fatal case is recorded by Mitchell.[2]

Sub-Group 3.—(1) Poisoning by Nitroglycerine ($C_3H_5(O\text{-}NO_2)_3$)

Source and Method of Occurrence. This is a sweet, oily, powerfully explosive liquid with a sweet aromatic pungent taste, well known to chemists as a substitution product of glycerine. It is much used in mining under the name of "blasting oil." Blasting gelatine is made by dissolving gun-cotton in nitroglycerine, and mixed with an infusorial earth it is known as dynamite. It is used in the manufacture of smokeless powders. Nitroglycerine in the shape of a 1 per cent. solution (liquor glycerylis trinitratis) is used in medicine as a vaso-dilator, official dose $\frac{1}{2}$ to 2 minims: it is an example of a class of drugs of which amyl-nitrite and nitrite of sodium are the best known members, but other organic nitrites such as erythrityl or erythrol tetranitrate are in common use.

Nitroglycerine is the most powerful of the known members of the group, and with the exception of amyl-nitrite the most rapid in its action. It is readily absorbed from the intact skin and has caused death accidentally in several instance.

The vapour of this liquid acts powerfully as a poison, and even when much diluted with air it produces intense headache.

These drugs all cause a rapid dilatation of the small arterioles, which lasts for a greater or less length of time, and their fatal effects are probably due to the consequent fall in blood-pressure and asphyxia from the conversion of hæmoglobin into methæmoglobin.

Duration. In fatal cases death is very rapid: thus a man drank some glonoin by mistake and died in three hours; and in other recorded cases only a few hours intervened between taking the fatal dose and death.

Symptoms. An overdose of nitroglycerine causes a throbbing headache and vertigo, the throbbing soon being felt over the whole body. The skin is cyanotic. There may be vomiting and diarrhœa. The breathing is dyspnoic. Convulsions may occur.

A man drank a considerable quantity of the liquid in mistake for beer. An hour later he was blue in the face and insensible. When admitted into hospital he was delirious and unconscious, and speedily became comatose. The hands were frequently raised to the head, as if there was headache. The face was red and swollen. He died six hours after swallowing the poison.

[1] R. Milbourne West, *B.M.J.*, 1896, 1, p. 146.
[2] *South African Medical Record*, 1912, 10, 276.

Honert met with a case of poisoning by this substance.[1] A man took a tablespoonful of gunpowder as a remedy for a boil, and, in order to increase its effect, added a few drops of nitroglycerine. Soon afterwards he was seized with great nausea, and violent and repeated vomiting. According to the patient's own account, he became black about the eyes, had extreme headache and giddiness, and several times became unconscious. There was intense vascular disturbance, and perspiration rolled in streams from the head and limbs. After some time the patient became paralysed; and four hours after the ingestion of the poison, the whole of the voluntary muscles, except those of the face and eyes, were paralysed. The pulse was thirty-nine, full and hard. The breathing became stertorous, and the extremities icy cold. In twenty hours the pulse had risen to seventy, then to ninety in the minute; and the paralysis disappeared. After an attack of catarrh of the stomach the patient quickly recovered.

A miner swallowed two mouthfuls. A painful feeling in his throat made him aware of his mistake, and he drank a quantity of milk. He was not seen by a medical man for an hour and a quarter. He was then suffering from faintness, difficulty of breathing, and oppression at the chest. In five hours vomiting and purging set in. Shortly before death the man lay quietly as if asleep, breathing feebly and occasionally with a deep sigh. The lips were livid before death.

Treatment. The patient must be kept recumbent with the head lowered. The stomach should be washed out if the poison was ingested. Adrenalin should be administered. Inhalation of oxygen and blood transfusion may be required to combat the methæmoglobinemia.

Post-mortem Appearances. These are not characteristic.

Analysis. Nitroglycerine is rapidly broken down in the body, and probably, in a case of poisoning, could only be isolated from stomach contents or vomitus, if at all. Even then the difficulties are very great owing to its close relationship to the fats. The material may be digested with methyl alcohol for about twenty-four hours filtered, the filtrate evaporated to a thick syrup and extracted with ether. This extract will suffice for a preliminary test, but may be purified by evaporation to a syrup, and extraction with cold alcohol, which will dissolve the nitroglycerine but leave most of the fat. Nitroglycerine is a heavy, oily-looking liquid. It is slightly soluble in water, more soluble in alcohol and ether. It explodes violently when struck or subjected to concussion. Nitroglycerine yields a red colour when treated with aniline and strong sulphuric acid, and also a red colour when treated with brucine and strong sulphuric acid (free from nitric acid).[2] When heated on the water-bath with yellow ammonium sulphide it is gradually decomposed to glycerol and ammonium nitrite.

(2) Poisoning by Cordite

Cordite consists roughly of about fifty-eight parts of nitroglycerine, thirty seven parts gun-cotton, and five parts of mineral jelly; while acetone is used as a solvent, but does not enter largely into the composition. Each cartridge contains sixty cylindrical strands of cordite, each strand measuring one and a quarter inches in

[1] *Deut. Klin.*, 1867, p. 83.
[2] *B.M.J.*, 2 : 925 (1903).

length, and one-twenty-fifth of an inch in thickness. It is no uncommon practice for troops to break a cartridge and chew a few strands of cordite. This produces all the effects of nitrite poisoning and the disorderly action of the heart may lead a young medical officer to grant a few days leave. By way of experiment Major Jennings, during the South African campaign, after discovering the existence of cordite-eating among a certain number of men, sucked a quarter of a strand for two minutes. He found that the diminution in its size after this time was scarcely appreciable, but nevertheless it caused the most racking, splitting headache he had ever felt in his life, which lasted quite thirty-six hours, and was accompanied by hammering and ringing noises in the ears. Its taste was sweet, pleasant, and pungent. From inquiries among the men, its effects appeared to differ somewhat when sucked and when taken in solution. Dissolved in tea, it produces an almost immediately exhilarating effect, "inciting to almost demoniacal actions." If many have partaken of the beverage all begin talking at once, each seemingly anxious to inform the other of everything that has happened to him since his birth. This condition is followed by heavy sleep and stupor, lasting five to twelve hours, according to the quantity taken. To awaken the subject it is often necessary to slap his face, punch or shake him, and awakening is accompanied by severe, dull, boring headache, muscular twitchings, and protrusion of the eyes. Even, however, when fully awakened, the cordite-eater does not seem to realise his surroundings for many hours. It is as an addition to beer that cordite appears to produce its worst effects. It then excites quarrelsome, destructive mania in an otherwise peacefully disposed individual, and produces immediate intoxication in a man who can commonly consume as much as four or five pints of beer without exhibiting a trace of having done so. If taken in quantity insufficient to produce sleep it makes him not only quarrelsome, but brings out the worst traits in his character. A possible clue to the inception of this habit is given by the fact that a large number of the men seem to have used cordite as a means of lighting pipes in default of matches. When thus used it gives a sweetish flavour to the tobacco, but causes great dryness of the throat, followed by headache. Others of the men seem to have first tasted the cordite out of curiosity. Major Jennings's account includes notes of communications made to him by various men, in all of which are found statements as to the exceedingly unpleasant after-effects. One of them said that he first became aware of this use of cordite from one of his comrades asking for something to straighten him up because he had been sucking it, and he also observed that some of the men became very elevated after taking a very small quantity of beer, though they could formerly take a large quantity of it without effect. "Cordite by itself," said the man, "does not seem to make men crazy, but only induces a very heavy sleep; but taken with beer or spirits, it brutalises the mildest man and makes a temporary maniac of him." He also added that he had noticed that it apparently aged a man rapidly, and made him extremely negligent of his personal appearance. The existence of the vice first became known to Major Jennings from the suggestion of a regimental non-commissioned officer that a certain man had possibly been taking cordite in order to escape service.

Mende[1] also records the use of picric acid by malingerers to simulate jaundice and avoid military service.

(3) Poisoning by Amyl-Nitrite

This is a volatile liquid substance and when inhaled produces all the effects of nitrite poisoning with great rapidity.

The following case of poisoning by ingestion of the drug has been reported :—[2]

A woman, aged fifty-eight, was under treatment for angina pectoris. She had from six to twelve attacks in the twenty-four hours. Nitrite of amyl inhaled (from capsules) quickly relieved the distressing symptoms. Owing to the costliness of capsules, a small quantity of amyl-nitrite was obtained, and this was given both internally and by inhalation, but had not the same effect as the capsules, so its use in this form was discontinued.

[1] *Deutsch. med. Woch.*, 44 : 1440 (1918).
[2] *B.M.J.*, 1 : 145 (1898).

On November 24th paraldehyde (two drachms) in water (two ounces) had been ordered. The nurse went to her locked cupboard and inadvertently measured amyl-nitrite, the two bottles (though not the labels) being exactly alike. It was only after she had given it to the patient that she realised from the odour and a remark of the patient what she had done ; the patient vomited within five minutes. Apomorphine gr. $\frac{1}{10}$, followed by cocaine gr. $\frac{1}{2}$, was given and she vomited freely ; hot water was given, and the vomiting encouraged. Half an hour after the pulse was weak, about 130 per minute, respiration shallow, with long intervals ; and temperature as low as 95°. The patient was semi-comatose, vomiting liquid smelling strongly of amyl-nitrite and having a brown colour. The pupils were contracted, and she was bathed in perspiration. Strychnine gr. $\frac{1}{30}$, followed by ergotinin cit., $\frac{1}{50}$, was given hypodermically, and whisky (two ounces) was given per rectum. The patient's breathing gradually became deeper and more regular, and in about two hours she was in her usual condition after an anginal attack.

Intense headache,[1] lasting some hours, as the result of inhaling the fumes of the drug is a common symptom which may be associated with throbbing of the vessels and dizziness.

Analysis. Amyl nitrite is a volatile liquid (B. pt. 96° C.) and may be separated—*e.g.*, from stomach contents—by distillation. The distillate, boiled with sodium hydroxide under a reflux condenser, is decomposed to amyl alcohol and sodium nitrite which may be tested for separately.

Sub-Group 4.—(1) Poisoning by Coal-Naphtha

Source and Method of Occurrence. The light oily product of the distillation of coal, a hydrocarbon known under the name of coal-naphtha, has caused death in one or two cases.

"A boy, *æt.* 12, swallowed inadvertently about three ounces of coal-naphtha. He soon appeared as if intoxicated, and ran about in a wild delirium. When seen in a short time by a medical man, he was insensible and collapsed, breathing stertorously, and his skin was cold and clammy. He had already vomited part of the liquid, and the odour of the vomited matter at once showed what he had taken. By the promotion of vomiting, he was made to eject altogether two table-spoonfuls of naphtha, and partially recovered. In spite of this reaction, however, in about two hours he was again in a state of collapse, insensible, pulseless, gasping for breath, and frothing at the mouth. The eyes were fixed and glassy, and the pupils contracted. There was complete loss of muscular power, and great difficulty of breathing, but no convulsions. He had lost the power of swallowing. In spite of every effort to save him, he died in less than three hours after swallowing the liquid. On inspection of the body four days after death a strong smell of naphtha was perceived throughout the tissues. The blood was fluid. There was slight effusion of serum in the ventricles of the brain. The right side of the heart contained fluid blood, the left was empty ; the lungs were not congested, but pale. The coats of the stomach were not inflamed nor materially changed in appearance. This organ contained a pint of semi-fluid matter, of which four or five ounces were liquid. An ounce of a dark-coloured liquid floated on the top, and was easily skimmed off. The liquid appeared to act in this case as a pure narcotic. There were no convulsions. The respiration of the *vapour* of this liquid diluted with air produces headache, giddiness, severe pain in the stomach, loss of appetite, and general illness."[1]

"On December 21st, 1900, a little girl aged five years, was taken to bed by her mother as usual at 6 p.m. The mother at the same time carried upstairs a pint bottle filled with coal-naphtha and placed it on the bedroom floor inside the door, intending to put it away in a cupboard at a more convenient opportunity. She then went downstairs ; at eight o'clock she again went up to the bedroom. Her attention was at once drawn to the little girl, who seemed to be breathing with extreme difficulty. Being alarmed at her condition, she cried out that the child was dying. Her husband immediately ran off to procure medical assistance. When I arrived at the house I found my patient in an almost comatose condition, breathing

[1] *Lancet*, 1856, 2, p. 230.

rapidly and with great difficulty. There was a strong, heavy, benzene-like odour in the breath which issued from the mouth and nostrils. The face was dusky and somewhat livid, and the hands were clammy and rather cold. The pupils were dilated, and the conjunctiva was insensitive to the touch. The pulse was rapid and small. On carefully examining the bottle which contained what remained of the liquid I concluded that the child must have taken from between two to three ounces. After injecting a small quantity of brandy with a glass syringe into the rectum the patient seemed to revive a little, and she vomited. A mixture of table-salt and warm water was then administered, which caused her to vomit freely three or four times. The vomited matter smelt strongly of the poisonous liquid, and consisted of partially digested food, together with a quantity of frothy mucus from the air-passages. The lower extremities of the child being next immersed in a foot-bath of hot water, into which a small quantity of mustard had been thrown, I flicked the face with a towel dipped in cold water, and also at regular intervals dashed cold water into her face. Artificial respiration was then performed, and when the livid discoloration had diminished to some extent, the body was inverted for a few minutes in the hope of exciting the respiratory centre in the medulla. External stimulants were also applied to the nostrils. The characteristic odour of the breath persisted for some time, which was possibly due in part to the fact that at the time of her imbibing the naphtha a certain quantity would doubtless have found its way through the glottis. The intensely laborious respiratory efforts, together with the presence of frothy mucus in the air-passages, may to some degree have been ascribed to the same cause. After the expiration of one and a half hours of continued and persistent application of restorative treatment, the child regained consciousness and began to cry. A teaspoonful of brandy in sweetened hot water was then administered, and I left the house. Later in the evening I learnt that she appeared to be going on all right and seemed somewhat inclined to sleep. On the 23rd, about 8 p.m., both parents called at my house and informed me that they thought the child had ' a touch of bronchitis.' I examined the patient the following day and found her to be suffering from acute general inflammation of the air-passages. The mouth and throat were likewise slightly inflamed, and an eruption—resembling herpes—had now broken out on the lips. The bowels being constipated, a dose of two teaspoonfuls of castor oil was prescribed. The chest was regularly poulticed ; demulcent drinks, such as milk and barley-water, etc., were ordered and brandy in teaspoonful doses every four hours was prescribed. The patient, however, gradually succumbed to the acute bronchitis dying at 1.15 a.m. on January 1st, 1901. An inquest followed in due course."[1]

Analysis. The peculiar odour as well as inflammability of the liquid, and the fact that it burns with a bright yellow smoky flame, would be sufficient to identify coal-naphtha. Its lightness and insolubility in water would allow of its being readily separated from the aqueous contents of the stomach.

(2) Poisoning by Benzene (Benzol, C_6H_6)

Source and Method of Occurrence. Benzene is a characteristically smelling liquid boiling at 80·5° C., and is obtained in enormous quantities from coal tar by fractional distillation, coming over first among the " light oils " up to 170° C.

Crude commercial benzene, 70°–140° C., contains, in addition to benzene proper, toluene (methylbenzene), xylene (dimethylbenzene), pseudocumene (trimethylbenzene), and other aromatic bodies. " Ninety per cent. benzene," so-called because in the distillation 90 per cent. comes over up to 100° C., contains 80–85 per cent. benzene, 13–15 per cent. toluene, and 2–3 per cent. xylene, and is used on a large scale in connection with the manufacture of dyes, synthetic pharmaceutical preparations, paints, etc.

There are many cases recorded of industrial poisoning by " benzene," especially in connection with the cleansing of the stills, and in the rubber

[1] *Lancet*, 1901, 1, p. 245.

trade. Accidental and suicidal poisoning are also known. Death usually occurs very quickly after a short illness, in which vertigo is a prominent symptom. Experimentally Rambousek showed that a concentration of ·056–·057 per 1,000 pure benzene in air causes almost instant symptoms in rabbits in the form of twitching of the muscles, convulsions in eight minutes, and coma within half an hour. Chloral hydrate checks the convulsions.

Toxicity and Fatal Dose. It acts as a narcotic and convulsant and causes destructive effects on the red cells and blood-forming tissues. The minimal lethal dose of benzene *per os* in man is not known accurately. In children 10–15 grams has proved fatal in minutes or hours. Spurr saw a girl of twenty die in fifty hours after ingestion of 30 grammes.

Symptoms. When the vapour is inhaled there is a feeling of dizziness and confusion, and consciousness is rapidly lost. Cyanosis and dyspnœa are usually pronounced. Exertion increases the severity of the poisoning, for in many cases the rescuer dies while the original victim recovers, although he may have inhaled the vapour for a much longer period. Concentrations of 5 parts per 1,000 will produce toxic symptoms in a few hours.[1] Air analyses in factories where cases of poisoning have occurred have given concentrations ranging from 1 in 5000 (0·7 mg. per litre) to 1 in 200 (17·5 mg. per litre).[2]

Chronic ill health may result from regular inhalation of benzene. The main effects are anæmia, the destruction involving both red and white cells so that the white count may fall below 1,000, and damage to the liver. There also appears to be a loss of certain substances necessary in coagulation of blood, so that hæmorrhages are common. The principal change found after death appears to be extensive hæmorrhages in the pleura and lungs, a change which has also been found in experimental benzene poisoning in animals.

In one of the cases reported by Jaffé a child of 1½ years swallowed some benzene while he was unattended. Almost at once he was seized with cyanosis, collapse, and vomiting of blood-stained slime, and died within 2½ hours. The *post-mortem* shows especially abundant hæmorrhages in both lungs, the hæmorrhages reaching up to the pleura. Microscopically the affected areas showed great dilatation of the capillaries and hæmorrhage into the alveoli. Gull observed a case of recovery from this poison.

In the *Lancet*, 1900, 2, p. 338, an inquest is reported on a boy who swallowed some ounces of benzoline. He speedily became unconscious, and died within thirty minutes. The case occurred at Burnham, Somerset.

A fatal case is also recorded by Dr. Spurr.[3]

Analysis. The odour, volatility, and inflammability of the liquid, as well as its insolubility in water, are sufficient to identify it, and to allow of its separation from organic liquids. The boiling point (80° C.) distinguishes it from its homologue, toluene (111° C.). By treatment with a mixture in equal volumes of strong sulphuric and nitric acids benzene is converted into nitrobenzene (*q.v.*, p. 622), which may be distinguished by its odour, resembling that of bitter almonds, and by various reactions.

[1] Greenburg, Public Heath Reports, Washington, 1926, 5, 41, 1257–75, 1410–31, 1516–[illegible]
[2] Methods for Detection of Toxic Gases in Industry No. 4, H.M. Stationery Office, 192[illegible]
[3] *Lancet*, 1899, 1, p. 1488.

Benzene in air may be detected and estimated by bubbling a measured volume of the air through a mixture of 10 c.c. of concentrated sulphuric acid and 0·5 c.c. of 40 per cent. formaldehyde. An orange-brown colour is produced. Toluene gives the same colour, as do coal-tar naphthas. Naphthalene vapour produces a black film on the surface of the reagent.[1]

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(3) Poisoning by Naphthalene (Naphthalin, $C_{10}H_8$)

Source. This substance is a solid obtained from the middle fraction in coal-tar distillation. It occurs in shining white crystalline plates with a characteristic odour. It is much used for disinfecting urinals, in " moth balls " and in the dye industry, etc. When moulded into blocks it is sold under several names—alabastrine, camphylene, albocarbon, etc.

Toxicity. Toxic symptoms have been produced by inhalation of the drug from sleeping under bed-clothes heavily dusted with the powder and also from ingestion of moth balls by children.

Such symptoms include nausea, vomiting, headache, profuse perspiration, jaundice and strictures hæmaturea. Experimental animals show inco-ordination, atoxia and partial paralysis. Cataract and other eye lesions may occur.

Evers[2] records the case of some chronic illness with loss of appetite, headache, and eczema over both legs, which was proved to be due to naphthalin poisoning. In this case the drug was used as moth powder, and was sprinkled over some bedding. The symptoms subsided directly the patient was removed from this particular room, but reappeared when the same apartment was reinhabited. No other cause for the illness could be discovered. Evers had the naphthalin examined and no impurity was detected. A case of typhoid is reported by Götze in which naphthalin was administered.[3] The patient was given six grams during the first three days; after this the dose was increased to seven grams. On the evening of the sixth day the patient began to be restless, and on the following evening he was delirious. The next day the patient was drowsy. The respiration was laboured and irregular. Lips and face cyanotic. Slight twitching in all the muscles of the body. Pulse regular, ninety-two per minute. The temperature had fallen to normal. The urine was dark brown, and after standing for some time became black. When naphthalin was discontinued the symptoms vanished in four days. Frommuller[4] saw three cases in which poisonous symptoms had followed the application of naphthalin to wounds. The symptoms began with a sudden onset of fever, headache, and loss of appetite.

[1] Methods for Detection of Toxic Gases in Industry, No. 4. H.M. Stationery Office, 1939.
[2] *Berl. klin. Woch.*, 1884, p. 593.
[3] *Ibid.*, 1884, 42.
[4] *Memorabilien*, 1883, 5, 257.

Zangerle[1] met with a case of naphthalin poisoning in his clinic at Marburg. A boy, aged twelve, came home one evening with symptoms which closely resembled alcoholic poisoning. The father was certainly of opinion that his child was "drunk." The patient was semi-conscious, his gait resembled that of a drunken man, he was unable to answer questions. The history of the case showed that no alcohol had been taken, but that a school friend had given him two "bonbons" which subsequently proved to be the cause of his illness. An emetic was given by the parents, which acted promptly. The next morning the author was called to see the boy, who appeared to be in a drowsy condition but was quite conscious; there was no vomiting, and the appetite had partly returned. The pulse was regular and full, the reflexes were lively, and there was no sign of paralysis nor incontinence of urine, also no discoloration of urine. The vomiting had so successfully cleared the child's stomach that it was thought unnecessary to give any medicine. The drowsy condition continued during the next four days, and then complete recovery took place. Another boy had taken one "bonbon," and had suffered from very similar symptoms, only in a less severe form. The offending sugar-plums were called "naphthalin camphor tablets"; they were white in appearance, resembling lumps of sugar. They were sold as moth destroyers.

Analysis. The odour is characteristic. Naphthalene may be isolated by steam distillation and extraction of the distillate with ether. It crystallises in glistening plates which melt at 80° C.

Chlorinated naphthalines are much used in industry as solvents for resins, waxes, etc. They produce varying degrees of acne and appear to have a toxic effect on the liver, and cause various systematic effects similar to naphthalene. Two fatal cases in workers have recently been described[2] in both of which extensive necrosis of the liver was found. Naphthol acts as a kidney irritant and nephritis may result from its absorption from the skin. Lesions of the lens and retina have been described.

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(4) Poisoning by Naphthol

Source and Method of Occurrence. From naphthalene, alpha-naphthol and beta-naphthol are prepared. When beta-naphthol is rubbed up with camphor it forms a viscid liquid known as naphthol camphor, described as a powerful non-toxic antiseptic for wounds.

Notwithstanding its non-toxic properties, at a meeting of the Surgical Society, M. Guinard reported the circumstances of a death which had occurred in his hospital practice very soon after an injection of twenty-five cubic centimetres of naphthol camphor. The patient was a man, twenty-eight years of age, who suffered from a chronic abscess in the right subclavian region. The puncture and injection were made in the usual manner and presented no special feature. The injection had scarcely been given when the patient had a sudden epileptic attack (*épilepsie généralisée*). Believing the symptoms to be of toxic origin, M. Guinard immediately made an incision and allowed the fluid to escape, but the patient nevertheless had two epileptic attacks and died in three-quarters of an hour. In addition to this case M. Guinard mentioned five others, of which two were unpublished and three have been published by M. Netter, M. Menard of Berck, and M. Estor of Montpellier, making a total of six deaths for which naphthol camphor was responsible. He also mentioned fourteen cases attended by symptoms of such severity that a fatal issue was apprehended. The pathogenesis of these conditions was very obscure. No objection could be raised either on the ground of the quality

[1] *Therap. Monats.*, February, 1899.

[2] *B.M.J.*, 1 : 691 (1942); *Lancet*, 1 : 72 (1943).

of the liquid, or the dose, or the age of the patients, or the operative manipulation. The only remaining possibility seemed to be a poisonous quality of the mixture of camphor and beta-naphthol, and M. Guinard has come to the conclusion that an injection of naphthol camphor properly administered in a small dose of five or ten cubic centimetres to a healthy adult or child might cause death in a few minutes.

Analysis. Beta-naphthol is excreted in the urine combined with glycuronic acid (as also is camphor). It can be recovered by distilling the urine with hydrochloric acid and extracting the distillate with ether. Dissolved in chloral, and warmed on the water-bath for a few minutes, beta-naphthol gives a blue colour, changed to yellow by HCl. Warmed with KOH solution and chloroform it gives a blue colour changing to green and finally to brown. (See also camphor).

(5) Poisoning by Iodoform (CHI_3)

Source and Method of Occurrence. This substance (in the form of yellowish crystals or powder) which has been used as an antiseptic in surgery, is without doubt a poison. As an antiseptic, it is inefficient, depending, apparently, upon slow decomposition with liberation of free iodine, and it is now seldom used. Schede, Kocher, and others have published cases where severe and even fatal symptoms have followed its external application. Boyd has reported four cases where toxic symptoms were developed after its use as a surgical dressing. These symptoms were, in two cases, drowsiness and stupor; in one, those of meningitis; and delirium in a fourth case, which terminated fatally.[2] Arbuthnot Lane has met with a case of poisoning by its local application to a wound.

Symptoms and Appearances. The symptoms usually observed after poisonous doses are—faintness, headache, giddiness, confusion of ideas, burning pain in the stomach, delirium, convulsions, insensibility, general paralysis, a small pulse, sometimes quickened and sometimes slowed, and the skin cold, livid, and bathed in perspiration.

A patient swallowed a quantity of iodoform that had been ordered for external application. Twenty-four hours afterwards he was attacked with violent headache, colic and diarrhœa which lasted through the following day. On the third day the pains disappeared, though the irritation remained. The breath, too, smelt of iodoform for several days, and a disagreeable taste remained in the mouth for a long time.[3]

Tuttle records the case of a woman who, twenty-four hours after using iodoform in her capacity of nurse, developed symptoms of poisoning by it. At first a very fine eruption of macules, papules and vesicles came out on the face, neck, hands and wrists; then followed a diffuse redness with œdema of the eyelids and backs of the hands; the conjunctivæ were intensely congested. The eruption appeared subsequently in all the parts exposed, but was most severe on the hands and between the fingers, where the vesicles became confluent, and, the epithelium having separated, a red oozing surface was left. On the fifth day there was some headache, itching was a prominent symptom, and desquamation took place on the backs of the hands.

Failure of vision (Brailey) is a common symptom of chronic iodoform poisoning.

Analysis. Iodoform may be separated from the material under examination by making alkaline with caustic soda, distilling in a current

[1] *Lancet*, 1904, 1, 1617.
[2] *B.M.J.*, 1882, 1, pp. 903, 913.
New York Med. Jour., 1891.

of steam, making the distillate alkaline, and extracting it with ether. The ethereal extract is allowed to evaporate spontaneously at a low temperature.

Tests. (1) Lemon-coloured hexagonal crystals, with characteristic smell; melts at 119° C. (2) Heated with a drop of aniline and a few c.c. of an alcoholic solution of KOH, it gives phenylisonitrile, recognised by its characteristic offensive smell. (3) An alcoholic solution, warmed with an aqueous solution of sodium phenate (phenol and sodium hydroxide) gives a red precipitate which dissolves in alcohol, giving a red solution decolorised by acid. This test and the preceding one are also given by chloroform. (4) Iodine can be liberated from iodoform by nitric acid or by fusion with NaOH, and extracted by carbon disulphide, which acquires a rose or violet-red colour.

Case. A man, aged thirty-five years, was admitted to the Swansea General Hospital, on March 5th, 1903, with a large non-tuberculous abscess on the calf and the thigh of the left leg. The temperature was 102° F., the pulse was 100, and the general condition was fair. On the 6th the temperature ranged from 98° to 99°. On the morning of the 7th the abscess was opened, the cavity irrigated, and a drainage-tube inserted. Whilst an assistant was putting in iodoform, quite half an ounce or more dropped accidentally into the large cavity. For twenty-four hours the temperature remained normal. At 8 p.m. on the 8th the patient had a rigor, when the temperature rose to 102°, and two hours afterwards it was 104°, the pulse being 140 and weak and thready and the respirations being 24 and normal in character. The patient, who now became restless and delirious but remained conscious, vomited three or four times. Nothing noteworthy regarding the pupils, the urine, etc., was observed. Quinine and brandy were now administered, and at 12 midnight, when the temperature was 103°, the cavity was washed and swabbed out, the iodoform forming quite a coat. In twelve hours the temperature, which had come down gradually, was normal, the pulse was 120 and fair, and the patient was quiet and sensible. This condition was maintained, the temperature remaining normal, the pulse being 80 and good, and the respirations being 24.[1]

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(6) Poisoning by Resorcin (Resorcinol)

Source and Method of Occurrence. Resorcin, or meta-dihydroxybenzene, is closely related to carbolic acid. It crystallises in colourless crystals, which are readily soluble in water, ether, or alcohol. Applied in the form of crystals, it acts as a powerful caustic to the skin. The commercial resorcin, which is obtained as a by-product in the manufacture of a pigment known as eosin, has a red colour, and a powerful odour resembling that of carbolic acid. It has occasionally been used internally as a medicine, but is more frequently employed as an application for the sake of its antiseptic qualities.

Of its **toxicity and fatal dose** but little is known from a medico-legal point of view. Resorcinol acts in much the same way as phenol, but appears to have a more convulsive action. It acts as a protein precipitant as does phenol, and has a similar caustic action on the skin. On one occasion

[1] *Lancet*, 1903, 1, 960.

in which an overdose of two drachms was given to a young woman, it produced insensibility, profuse perspiration, blanched lips, equal normal pupils, lowness of body temperature, imperceptible pulse, and almost imperceptible breathing. The urine was olive-green in colour. The patient recovered.[1]

Analysis. Ferric chloride produces a violet colour; sulphovanadic acid, blue and then violet. If a crystal of sodium nitrite is mixed with a drop or two of concentrated sulphuric acid and a little resorcin is added, a violet colour, which changes to blue and then brown, is produced.

If a fragment of resorcin is fused with phthalic anhydride and dissolved in dilute caustic soda a yellowish green fluorescent odour is produced.

Case. Schwabe reports a case of poisoning by resorcin. A girl, aged five years, was ordered an intestinal irrigation of 100 grams of a ½ per cent. solution of resorcin for gastro-enteritis. By mistake 200 grams were used (one gramme of resorcin). A few minutes afterwards the child became pale, with clammy perspiration, retraction of the head, and muscular stiffness. She soon became unconscious and pulseless in fifteen minutes. Medical aid was at once obtained, and ether administered subcutaneously; the colon was washed out with sulphate of soda solution. After ten minutes consciousness was regained, and the pulse returned. Stimulant treatment was continued, and the child gradually recovered, but was very weak. The urine was dark green and albuminous. The child recovered within a week.

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(7) Poisoning by Formaldehyde

Source and Method of Occurrence. Formaldehyde itself is a colourless gas, with a powerful irritating odour. It is generally used as a 40 per cent. solution in water; it is then termed formalin, and is used extensively as a preservative in pathological museums. It is largely used in industry in the manufacture of "bakelite" and other plastics, artificial horn, etc., in the hardening of celluloid, and as a reducing agent. Formalin at ordinary temperatures gives off a vapour of the aldehyde, which is most irritating to the eyes, mouth, and bronchial tubes, and may cause bronchitis and pneumonia. It causes most unpleasant effects upon the hands when these come in contact with the material.

If it is swallowed it causes acute gastric irritation with severe immediate abdominal pain, prostration, loss of consciousness and death within 24 to 48 hours.

In most fatal cases the kidneys are damaged, and after a period of anuria there may be blood and casts in the urine. Diarrhœa may occur.

Treatment. Gastric lavage with administration of sodium bicarbonate or sodium phosphate.

Case. Not many cases have been recorded of fatal poisoning by it, but the following is taken from the *Pharm. Journ.*, September 3rd, 1899:

A young man drank about two ounces of a 4 per cent. formaldehyde solution which was used for treating seed potatoes. The immediate effect was to cause vomiting of matter containing traces of blood, and death occurred about twenty-nine hours afterwards, as the result of heart failure. A *post-mortem* examination showed that the œsophagus was slightly inflammed, and escharotic changes were visible in the stomach.[2]

[1] *Med. Times and Gaz.*, 1881, 2, p. 487.
[2] *Vide* also *B.M.J.*, Epit., 1901, 1, p. 72.

In the *B.M.J.*, Epit., 1901, 1, Nos. 42 and 300, will be found other cases of poisoning by this substance.

Analysis. Apart from the characteristic odour, formaldehyde may be detected in very dilute aqueous solution by the usual aldehyde tests. It reduces ammoniacal silver nitrate. It restores the colour of Schiff's reagent. With alcohol and a few drops of diphenylene dihydrazine it gives, on warming, a yellow solution which deposits a yellow crystalline precipitate after standing for some time. (See also methyl alcohol.) Owing to its volatility an aqueous distillate suitable for the application of the tests is readily obtained from neutral or slightly acid organic fluids. The urine may contain formic acid.

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Sub-Group 5.—(1) Poisoning by Aniline ($C_6H_5NH_2$)

Source and Method of Occurrence. Aniline (phenylamine), or aniline oil is a derivative of coal tar. It is an almost colourless liquid, mobile and oily, with a faint vinous odour and aromatic burning taste. It is prepared in enormous quantities for use in industry. Commercial " aniline oil " usually contains a number of impurities in small amount — *e.g.*, toluidine, nitrobenzene, etc.

It is occasionally used for suicidal purposes, accidents frequently occur among those working with it, but it is rarely if ever used for homicidal purposes.

Cases of poisoning from the cutaneous absorption of aniline are the most common.

Muehlberger [1] has described several cases of poisoning from shoe dyes in which the solvent used was aniline. The shoes were worn before the dye was quite dry, but no staining of the feet took place. Intense cyanosis, headache, vertigo, and weakness were the main symptoms observed in printers and rubber workers.

Cases have arisen in painters using aniline-containing paint ; from the use of hair dyes ; from the absorption of marking ink, etc.

Toxicity and Fatal Dose. Death has followed the ingestion of 25 c.c. of aniline ; on the other hand, a dose of 75 c.c. has been survived.

Turnbull gave half a drachm of the sulphate to a dog. In two hours and a half the animal vomited, and an hour later it was purged. It became dull, weak, and tremulous ; the pulse was rapid, and the breathing laboured. The feet were cold, the hind legs paralysed, and the tongue was of a blue colour. In five hours the symptoms abated, and the next day the animal had recovered.[2]

Letheby found that aniline given to dogs and cats, in doses of from twenty to sixty drops, caused a rapid loss of voluntary power. The animal staggered and fell upon its side powerless, the head was drawn back, the pupils were dilated, the breathing was difficult, and the action of the heart tumultuous ; there were slight twitchings or spasms of the muscles, and the animal quickly passed into a state of coma, from which it did not recover, death taking place in from half an hour to thirty-two hours. On inspection, the brain and its membranes were congested, the cavities of the heart were nearly full of blood, and the lungs but slightly congested. The blood all over the body was black and coagulated.

[1] *Jour. Amer. Med. Assoc.*, June 27th, 1925.
[2] *Lancet*, 1861, 2, p. 469.

Duration. Recorded cases seem to show that there is usually some delay in the onset of symptoms.

Symptoms. These are commonly of a narcotising type, like those of nitrobenzene, commencing with weakness in the limbs, staggering gait, nausea, headache, dizziness and roaring in the ears. The heart and respiration rates are usually increased. Mental confusion and amnesia may occur. A typical feature is the cyanosis which is caused by the absorption into the blood of aniline ; this is most marked about the lips and finger nails, and in fatal cases can be recognised in the blood as a bluish discoloration. The colour of the skin and blood, which is such a prominent sign of aniline poisoning, is often stated to be due to methæmoglobin produced by the destructive action of the drug on the red cells. This is denied by many investigators ; others, however, claim to have detected the methæmoglobin spectroscopically, and in one case its gradual disappearance with repeated blood transfusion is described. It is quite possible that the pigmentation may be partly due to oxidation products of aniline. Jaundice has also been described in more prolonged cases. In treatment, oxygen inhalations appear to be of little use, and as a rule a long period elapses before the normal colour returns. The colour is lilac or bluish in light poisoning, but in severe cases it changes to a deep blue or sometimes a slaty grey.

Rehn has drawn attention to the occurrence of tumours of the bladder in aniline workers, and Bachfeld saw sixteen bladder affections among sixty-three cases of aniline poisoning. Observations in eighteen aniline factories showed thirty-eight cases of tumours of the bladder, of which eighteen terminated fatally. Curschmann, in 1920, gave a list of 177 cases.[1]

Kreuser has noticed among the workers in aniline that they have suffered from intense bronchitis, with a violent dry spasmodic cough, accompanied by ulcerations on the scrotum and extremities. Anæmia, various nervous symptoms and skin eruptions, have also been described.

A workman accidentally broke a carboy containing a large quantity of this liquid ; the aniline fell over him, but none entered his mouth. In his anxiety to wipe up the aniline, he respired the vapour for some time, felt giddy, and complained of his head and chest. When seen some hours after the accident, his face and body were of a livid leaden hue, the lips, gums, tongue and eyes of a corpse-like bluish pallor ; his breathing was gasping, and he appeared at the point of death. There was no convulsion ; he was sensible and able to give an account of his feelings. His pulse was small and irregular. Under active treatment he recovered.[2]

A boy, *æt.* 16, was brought into the London Hospital in a semi-comatose condition. In scrubbing out an aniline vat he had breathed the vapour ; and although he did not suffer pain or discomfort at the time, he was suddenly seized with giddiness and insensibility. When brought to the hospital he looked like a person in the last stage of intoxication ; the face and surface of the body were cold, and the pulse was slow and almost imperceptible, the action of the heart was feeble, and the breathing heavy and laborious. After rallying a little, he complained of pain in his head and giddiness. His face had a purple hue, and his lips, the lining membrane of his mouth, as well as his nails, had a similar purple tint. On the next day the narcotic symptoms had passed away, but he was remarkably blue, and looked like a patient in the last stage of Asiatic cholera.[3]

[1] *Z. Gewebehyg.*, 1920, 8, 145, 169.
[2] *Pharm. Jour.*, July 1862, p. 42 ; *Med. Times and Gaz.*, 1862, 1, p. 583.
[3] *Med. Times and Gaz.*, 1862, 1, p. 239.

These cases appear to show that aniline vapour is less poisonous than that of nitrobenzene, and that the symptoms follow more rapidly on the inhalation of the vapour.

Some of the aniline dyes used for marking or dyeing underlinen have produced much irritation and sometimes an eczema by contact with the skin.

Treatment. Empty the stomach as quickly as possible with a solution of magnesium sulphate ; give stimulants ; oxygen inhalations may be tried, but they rarely prove serviceable. Artificial respiration may be necessary. If aniline has been spilt on the skin, it must be washed off immediately.

Post-mortem Appearances. The peculiar colour of the blood is very characteristic ; beyond this there is nothing to see.

Analysis. *Commercial* aniline is an oily liquid of a reddish-brown colour, with a peculiar tarry odour. It produces a volatile greasy stain on paper. It is volatile and combustible, burning with a thick, smoky flame. It falls to the bottom of water and does not readily dissolve in it. It is soluble in alcohol and ether, but only sparingly in chloroform : in the latter property it differs from nitrobenzene. Sulphuric acid combines with it to produce a white sulphate, soluble in water. A solution of chlorinated lime added to the acid watery liquid produces a blue colour, passing into various shades of purple and brown. If a few drops only of a very dilute solution of aniline or its salts be warmed in a test-tube with alcohol, caustic potash and a drop or two of chloroform, the unpleasant odour of phenyl isonitrile is evolved. A trace of aniline, dissolved in concentrated sulphuric acid and treated with a little potassium bichromatic solution, gives a blue colour which develops slowly, persists for some time and then fades without changing to purple and red (*cf.* strychnine). If more water is present, the colour may be green.

The solution of sulphate of aniline is not precipitated either by tannic acid or by chlor-iodide of mercury and potassium ; but aniline itself, in the small quantity in which it is dissolved by water, yields, like the alkalies, a yellow precipitate with arsenio-nitrate of silver. It also reduces completely a solution of auric chloride—precipitating metallic gold. When pure aniline is heated with powdered corrosive sublimate, it produces a rich crimson dye. When present in organic liquids, aniline may be separated by distillation from an alkaline medium in a current of steam.

Dr. St. Clair Thomson[1] read a paper before the Clinical Society on a case of "Poisoning from the External Use of Aniline Oil." Equal parts of aniline oil and rectified spirits having been recommended as a vehicle for cocaine in order to produce local anæsthesia in the ear, Dr. Thomson prescribed a 10 per cent. solution of cocaine in this menstruum for a colleague suffering from furunculosis. A small pledget of cotton-wool moistened with this solution was used at bedtime, and the patient slept well. Next morning, as the pain threatened to return, he again made use of the drops about 5 a.m. At 7.30 a.m., while still in bed, he quite accidentally noticed a peculiar blueness of his finger nails, and his wife remarked that his face was also blue. The face and hands were found to be of a decided dark blue colour, and this was noticeable in the skin under the finger nails and on the lips and tongue. There was no fever nor mental disturbance. The pupils were normal. The respiration was quiet and easy. The pulse was small and somewhat increased in frequency. Recovery soon followed on discontinuing the remedy.

Lancet, 1 : 1143 (1901).

Reference was made to a communication made to the Académie de Médecine in July last by M. Landouzy and M. Georges Brouardel describing the cases of ten children who were seized with prostration, pallor, and blueness soon after wearing yellow shoes which had recently been coated with a pigment found to contain 90 per cent. of aniline. When this substance was applied to the shaven surface of the skin of guinea-pigs and rabbits they died asphyxiated in from twenty-four to thirty hours. Some unpublished cases of Dr. Kelynack described similar symptoms together with gastro-intestinal catarrh and anæmia in chronic cases, among those employed in aniline works.

Dr. John Inkster has recorded the following non-fatal case [1] :

A lesson in distillation was being given to a chemistry class, aniline being the substance used to demonstrate this process. As the aniline was to be returned to stock after distillation, the master told the boys to be careful that no water was mixed with it. One boy (*æt.* 12) distilled his portion into a wet flask, and when he handed it to the master, the latter said, in a jocular manner, " This is a nice mess. It is not fit to be returned to the bottle. You might as well drink it." This the boy proceeded to do. The master immediately dashed the flask from the boy's hand and made him wash out his mouth. The flask contained less than 2 c.cm. of a mixture of aniline and water, and the master was under the impression that the boy had not swallowed any of it.

The boy carried on at school from 9.45 a.m., the time of the incident, and made no complaint nor did anyone notice anything amiss. At 12.20 he took train to his home and arrived at his station without mishap but collapsed outside at about 12.45, and was carried home. He was then unconscious, the finger-tips were blue and the lips almost black. Oxygen was administered continuously through a funnel from about 1 o'clock and coffee and whisky given frequently from then until 4 o'clock. He vomited repeatedly from 2.30 onwards and at 3 o'clock was roused enough to say that he had taken aniline but immediately relapsed into unconsciousness.

Seen at 10 p.m. he was lying peacefully in bed with his eyes shut. He was very drowsy but could be roused with difficulty when he would open his eyes, but would not answer questions. The skin was a pale lilac colour all over except that the lips, ears, tongue, and the nails of the fingers and toes were of a deep leaden blue. The respirations were slow and very shallow. There was no apparent respiratory distress. The pulse-rate was 120 and the pulse regular and small. There was no dilatation of the heart and no abnormal signs were detected in the lungs. The pupils were widely dilated and reacted very sluggishly to light. There was no injection of the conjunctivæ. There was no paralysis. The tendon reflexes were somewhat diminished.

As it was so long since the ingestion of the poison and he had vomited repeatedly, there seemed to be no point in washing out the stomach. Oxygen was given through a nitrous oxide bag for five minutes at a time with intervals of 15 minutes instead of constantly through a funnel. Very soon there was improvement. The patient became less drowsy and began to resist the application of the face-piece of the gas-bag until consciousness was restored sufficiently for him to be reasoned with, after which he took the oxygen well. After about three-quarters of an hour he had recovered sufficiently to sit up and take some tea, the dilatation of the pupils had become much less and the colour had improved greatly. He was then given mag. sulph. ℥ii with tr. belladonnæ ♏v and water to ℥ii. Oxygen inhalations were continued until noon the following day when they were no longer necessary. The mag. sulph. mixture was repeated twice on successive days.

Progress was uneventful. He felt no ill-effects except headache, which persisted for three days. The blueness gradually cleared off, and was completely gone after three days.

Three weeks later he felt quite well and showed no sign of any ill effects. He stated that he had less than a teaspoonful of the mixture of aniline and water in his mouth, and thought that he swallowed some of it but was not sure how much. After taking it he noticed a burning sensation in his mouth and immediately began to feel dizzy. The dizziness increased but he was able to go on with his work. Just before he got on the train on his way home, two and a half hours after taking the aniline, he noticed that his legs were becoming weak and that the dizziness was much worse.

[1] *Lancet*, October 9th, 1926.

(2) Poisoning by Nitrobenzene, or Nitrobenzol, Oil of Mirbane ($C_6H_5NO_2$)

Source and Method of Occurrence. This liquid has a smell resembling that of benzaldehyde (or oil of bitter almonds sine acido-hydrocyanico), but the two are very different in other respects, and must not be confounded with one another, though nitrobenzol is used as a substitute for benzaldehyde in cheap perfumery and soaps. It is also used very largely in the production of the aniline colours, as it is converted into aniline by reduction—*e.g.*, with hydrochloric acid and iron filings. Grandhomme, who studied the health of the workers at the great factory at Hochst, could find no case of poisoning among twenty-one men employed in making nitrobenzene, although some had been working from ten to twenty years. Acute and chronic poisoning, however, may occur from accident or carelessness.

A good many cases of poisoning have occurred from the use of shoe dyes in which nitrobenzol or aniline were used as dye solvents. Muehlberger has listed forty-seven such cases [1], and Stipel has described seventeen cases which occurred in soldiers who had their shoes dyed to match their puttees.[2]

It does not appear to have been used homicidally, possibly on account of its nauseous taste and extremely persistent smell, but accident and suicide account for a number of cases; five cases published by Von Buschow were due to drinking brandy with an uncertain admixture of it,[3] two fatal and three ending in recovery. In the Franco-Prussian War (1870–71) Helbig observed an epidemic of eighteen cases with three deaths in soldiers from drinking nitrobenzol under the impression it was a liqueur.

Toxicity and Fatal Dose. The liquid is very poisonous; twenty drops have been known to cause death, but recovery has taken place after much larger doses.

It is absorbed through the skin with great ease and rapidity, and gives rise to severe intoxication.

In performing some experiments on animals, Letheby found that the local action on the stomach was slight; there was rarely any vomiting, and there was either rapid coma, or a slow setting-in of paralysis and coma, after a long period of inaction. There was a complete loss of voluntary power, a spasmodic fixing of the muscles of the back, with violent struggles, a look of distress, and occasionally a kind of epileptic fit. The pupils were widely dilated, the action of the heart was irregular, and the breathing difficult.

Duration. When the poison is taken by the mouth, the onset of symptoms is often delayed for an hour or more, and death rarely takes place for several hours. In these respects it strongly contrasts with hydrocyanic acid, which its smell suggests. Recovery is very slow (*vide infra*). The time of death in animal experiments varied from twenty-five minutes to twelve hours after the administration of the poison. In other experiments, in which smaller doses were given, the time that elapsed between the administration of the poison and the onset of symptoms (an epileptic fit) varied from nineteen to seventy-two hours;

[1] *Jour. Amer. Med. Ass.*, 84 : 1987 (1925).
[2] *J. A.M. A.*, 72 : 395 (1919).
[3] *Berl. klin. Woch.*, March 4th, 1895.

in most cases it was about two days, and the time of death was from four to nine days. In man death has occurred as late as the seventeenth day.

When the poison is absorbed *viâ* the skin or lungs, the symptoms appear much more rapidly and death may take place within an hour.

Symptoms. The usual symptoms are severe headache, nausea and vomiting, rapidly succeeded or preceded by faintness and unconsciousness; skin irritation, muscular twitchings and unsteady gait are sometimes observed; vertigo is common, and extreme cyanosis is the most marked feature, extending to the finger nails. The drug acts upon the blood, altering the hæmoglobin into a pigment which is useless for oxygen transmission. This pigment is sometimes stated to be methæmoglobin, but there is considerable doubt whether this is the main pigment produced. According to Young and others,[1] the brownish discoloration of the blood and urine is due to the presence of *p*-aminophenol. The red cells undergo rapid destruction and the urine contains various hæmoglobin derivatives, albumen and casts. The motory cells of the cutex are probably directly affected and convulsions followed by coma and death within a few hours may occur. The following cases illustrate the above statements; they tend to show that the vapour is much more potent than the liquid.

Nicholson,[2] in referring to a fatal case of poisoning by the liquid, states that he has known several instances in which the *vapour*, as it is evolved from almond glycerine soap, has seriously affected persons. A friend of his who used a cake of the soap in taking a warm bath fainted from the effects of the vapour of nitrobenzene set free, and was ill for some time afterwards. In 1863, a case of poisoning by this compound occurred, in which the symptoms so closely resembled those of essential oil of bitter almonds that it was at first supposed this oil had been taken. A woman, *æt.* 30, tasted a liquid which had been used for flavouring pastry, and perceiving that it was very acrid on her tongue and lips, spat it out immediately and washed her mouth with water. She thought she could not have swallowed more than a drop, but in replacing the bottle she spilled about a tablespoonful on the table and did not immediately wipe it up. The vapour strongly impregnated the small room in which she was, and produced a feeling of sickness in another servant. The burning taste in the mouth was immediately followed by a sensation of numbness and tingling in the tongue and lips, and a strange feeling for the next hour. The woman became worse, and Fotherby saw her in an hour and three-quarters after the occurrence. Her aspect was then quite like that of prussic acid poisoning: the eyes were bright and glassy, the features pale and ghastly, the lips and nails purple, as if stained by blackberries; the skin was clammy and the pulse feeble. Her mind was then clear, and she described how the accident had occurred, and what her sensations were. She was able to swallow a mustard emetic, after which she became rapidly worse, consciousness was lost, the teeth became set, the hands clenched and blue, the muscles rigid and convulsed. She vomited freely a pale fluid matter, which had the odour of nitrobenzene. The stomach-pump was used, but the fluid washed out of the organ had hardly any odour. The breathing became much reduced, and the pulse could scarcely be felt. In about eleven hours there was reaction, consciousness returned, and she was able to swallow. At the end of seventeen hours she was much better; but she then complained of distorted vision, with flashes of light and strange colours before her eyes. For some weeks she continued weak. It was at first supposed the woman had swallowed a larger quantity of the liquid than she had imagined; but it is obvious, from the entire absence of the odour in the fluid drawn off by the stomach-pump, within about two hours, that but little could have passed into the stomach. There is no doubt, from what has been observed in other cases, that these severe symptoms were chiefly due to the breathing of the vapour in a concentrated form. A fellow servant

[1] *Jour. Pharm. and Exp. Therap.*, Baltimore, March 27th, 1926.
[2] *Lancet*, 1862, 1, p. 135.

who was in the room at the time the liquid was spilled also suffered from the inhalation of the vapour. The liquid was found to be nitrobenzene unmixed with essential oil of almonds.

A clerk in some chemical works took a few drops (supposed to have been fifteen) of nitrobenzene. Immediately afterwards he felt unwell and became insensible. Stimulants restored consciousness, but there was a relapse, and he died the next day.[1] A boy, *æt.* 17, while drawing off some nitrobenzene by a syphon, swallowed a portion of the liquid. There were no immediate symptoms, but he soon felt sleepy, and when at dinner ate but little, and said he felt as if he were drunk. This was between two and three hours after he had swallowed the liquid. He fell into a stupor which became deeper and deeper until death took place, without vomiting or convulsions, twelve hours after the ingestion of the poison.[2]

A case is reported of a man who took a dose of furniture cream by accident whereby he ingested approximately 15 minims of nitrobenzene. Beyond slight vomiting no symptoms occurred for several hours. He then became drowsy and markedly cyanosed. After gastric lavage and blood transfusion he gradually made a complete recovery.[3]

Treatment. Removal of the source of the poison is the first essential, whether it is being absorbed from the skin, the air, or from the alimentary canal. The stomach must be emptied by the stomach-tube or emetics as rapidly as possible, and thoroughly washed out with magnesium sulphate. Cold douches and other stimulants may be employed to rouse the patient and prevent stupor. Artificial respiration should be tried. Bleeding and transfusion may be useful in bad cases.

Post-mortem Appearances. Nitrobenzol seems to be partially converted into aniline in the body, and this latter body has a peculiar power of turning the blood a bluish colour. In addition there is usually a moderate amount of jaundice and the destruction of red blood corpuscles may be intense. The discoloration and the powerful smell are the most characteristic features observed on *post-mortem* examination. There may, however, be degenerative changes in the heart muscle, the liver and kidney and hæmorrhages into the serous membranes. Sometimes the liver suggests acute atrophy. When death had taken place within twenty-four hours, the odour of the nitrobenzene was clearly perceptible in the stomach, brain, and lungs; and aniline (from the chemical conversion of nitrobenzene) was found in the organs. In the slower fatal cases the odour had often entirely disappeared; but traces of aniline could be detected in the brain and urine, and sometimes in the stomach and liver. Occasionally no trace of the substance was found, although death had taken place from the poison. Para-aminophenol may be found in the urine (for test, *vide* p. 585), and methæmoglobin may be detected spectroscopically in the blood.

Analysis. Nitrobenzene, or "essence of mirbane," is a pale lemon-coloured liquid, with a strong odour resembling that of bitter almonds. It has a pungent, hot, disagreeable taste. It gives to confectionery the smell, but not the pleasant taste, of oil of bitter almonds. It gives a greasy stain to paper, leaving a yellow mark when the stain disappears. It sinks in water, and is slightly dissolved, giving a yellowish solution. It is soluble in alcohol, ether, and chloroform: but when these are agitated with water, it is in great part separated from its ethereal and

[1] *Pharm. Jour.*, December, 1862, p. 283.
[2] *Med. Times and Gaz.*, 1862, 1, p. 239.
[3] *B.M.J.*, 1 : 557 (1945).

chloroform solutions. It has no basic qualities; its aqueous solution is not precipitated by tannic acid nor by the chloriodide of mercury and potassium. It is highly combustible, burning with a yellow smoky flame. It yields no Prussian blue when mixed with ferrous sulphate, alcohol, potash, and subsequently hydrochloric acid; and its vapour produces no cyanide of silver with a solution of the nitrate. It is distinguished from all other liquids, except the essential oil of almonds, by its odour, and from this oil by the following test: Pour a few drops of each on a plate and add a drop of strong sulphuric acid; the oil of almonds acquires a rich crimson colour with a yellow border, the nitrobenzene produces no colour. With a crystal of chlorate of potassium and a drop of sulphuric acid, it yields a violet coloration. In order to separate it from organic liquids they may be acidified with sulphuric acid, and submitted to distillation in a current of steam. If any of it exists in a free state its odour will be sufficient for detecting its presence. It is converted into aniline by acting upon it with hydrochloric acid and zinc dust. After the liquid has been made alkaline with sodium hydroxide the aniline may be subsequently distilled over with steam or extracted with ether and identified in the aqueous distillate or the ethereal extract by the tests for aniline (*q.v.*, p. 620).

Poisoning by Roburite and Bellite

Roburite and Bellite both contain dinitrobenzene and ammonium nitrate. They are present in safety explosives, and have often caused poisoning. In the Witten roburite factory from 1890–7 there was a high incidence of illness among the workers, the majority of whom showed pallor, blue lips and yellowish conjunctivæ.[1]

Analysis. Dinitrobenzenes are reduced to phenylenediamines by reduction (tin or zinc and hydrochloric acid). After separation from organic matter by steam distillation, this reduction may be carried out on the distillate. The liquid may then be made alkaline and extracted with ether, which dissolves out the metaphenylene diamine. Tests for this substance are given on p. 628.

Poisoning by Phenylenediamine

Two phenylenediamines (meta and para) are important dye intermediates, and they or certain of their oxidation products have a definitely toxic effect on the skin causing a persistent dermatitis. After absorption, these substances cause tremors and other nervous disturbances, dizziness and headache. There may be marked muscular weakness, and in severe cases convulsions, coma and death.

Inhalation of the drug sometimes causes an acute attack of asthma or bronchitis, with sneezing and great depression.

The substances are used not only in dyeing furs, but also for hair-dyes, and the practitioner should be on his guard when confronted with cases of dermatitis with or without general symptoms, which have arisen without apparent cause.

[1] Schröder und Strassmann. *Vierteljahrsschr. f. ger. Med.*, 1891, Suppl., 138; Brat, *Deutsche med. Woch.*, 1901, No. 19 20. Also *B.M.J.*, 1895, 1, p. 39, a fatal case of poisoning by bellite; and *Lancet*, 1901, 2, p. 1613, one by roburite used as an insect powder.

It appears that when either of the phenylenediamines is treated with an oxidising agent, two substances are formed, one of which is soluble and irritating, the other insoluble and harmless. The fur after dyeing must be thoroughly washed to get rid of the former product, otherwise on wearing the articles, dermatitis is produced.

The Ministry of Health has issued a report on this matter.[1]

Jacoby[2] records the following case of fur dermatitis :—

A woman, aged twenty-nine, who had no previous skin trouble, purchased a coat with a fur collar late in 1923. Almost immediately after wearing it for the first time she felt an itching sensation, and an intensely pruritic reddish rash appeared on the left side of her neck. The eruption subsided in a few days during which the coat was not worn. Four days later she began to wear the coat daily, and the skin lesions became aggravated and spread to the chest, arms, forehead, brows, and lips. Seven weeks later there was an erythematous eruption which involved the entire neck and extended slightly above the hair line ; it involved also the chin, face, and chest and the flexor aspect of the forearms ; many vesicles and crusts were present. She ceased to wear the coat, and the lesions disappeared almost completely after three weeks. A few weeks later the coat was again used, and even more severe lesions of an eczematoid character appeared in the same situations. Paraphenylenediamine was found in the fur ; no arsenic was present.

Nott[3] records the following case of poisoning from phenylenediamine hair dye.

The proprietor of a hairdressing saloon, of middle age, looking apparently well and who presented no physical signs of disease, came for examination on account of serious and unaccountable illness, which as a rule came on suddenly. On one occasions when he was preparing for bed an attack came so suddenly and with such tumultuous action of the heart that he fell on the bed helpless, certain he was dying, and lapsed into unconsciousness, from which he must have passed into natural sleep, for he awoke next morning perfectly well, to find himself lying across the bed half dressed.

A week later he telephoned at 9 a.m. saying he had gone to bed perfectly well the night before and had wakened that morning to find himself in for another attack. When seen at 3 p.m. the same day he at first sight gave the impression of one who might have had several nights' drunken debauch, but his breath was perfectly healthy. His face was grey, cyanosed, and blotchy. His eyes were watery, the conjunctivæ injected, and the eyelids swollen. The lips and ears were violet, the tongue was swollen and covered with a thick yellow and brown fur. The gums were very swollen, standing away from the teeth, and purple. Some of his fingers were stained, apparently with iodine. The pulse was 105, regular, and fairly strong ; the temperature was normal.

He was nauseated, and had taken no food all day. He complained of pain, chiefly in the epigastric and hypochondriac regions, and between the shoulders. There was slight palpitation, and he looked anxious and strained. He described some difficulty in swallowing, with a burning sensation in the throat and behind the sternum. When he took a deep inspiration he felt severe pain in the chest, and a choking sensation. As the previous day, Sunday, had been spent in the house, and as all food taken had been prepared at home, it was impossible to resist thoughts of secret poisoning by arsenic, for he somewhat resembled a series of cases seen here twenty years ago when arsenic accidentally contaminated the beer from several breweries in the north of England.

When it was explained that his symptoms resembled some forms of poisoning, and when he began to think of the possibility of some chance method of poisoning, he supplied the clue himself by asking if hair dye could do it, for it was about three years ago when he first used it. He often worked in the fumes of it, and occasionally applied the dye to his customers without wearing gloves which accounted for the stains on his fingers—the last occasion being about forty-eight hours before this interview.

[1] See *Lancet*, September 6th, 1924.
[2] *Boston Med. and Surg. Jour.*, April 1925, p. 852.
[3] *B.M.J.*, March 8th, 1924.

Two bottles of the dye were sent to an analyst with a note that it was under suspicion for causing mixed symptoms of lead and arsenic poisoning in a man who worked with it, and the following report was received :—

" In accordance with your instructions we have examined the two solutions of hair dye which you handed to us.

" One solution gives the reaction for both metaphenylenediamine and paraphenylenediamine ; the other is a solution of hydrogen peroxide.

" Paraphenylenediamine is commonly used as a hair dye, and injurious effects on workpeople engaged in the manufacture of this compound are attributed to the diamine itself and to its oxidation products.

" Metaphenylenediamine possesses marked poisonous properties, its physiological action resembling that of the leucomaines and ptomaines. An authority states that a dose of 0·1 gramme per kilo body weight produced salivation, vomiting, diarrhœa, and abundant excretion of urine in dogs and subsequent death by coma. Symptoms similar to those of intense influenza were also produced in addition to the more severe effects.

" Phenylenediamine derivatives are used in the production of dyes for furs, and are commonly used in the preparation of brown dyes."

Three months have elapsed since the report was received, and by avoiding exposure to further risk of absorption, the patient has had no return of his illness.

The case adds further evidence of the peculiarly selective action of phenylenediamine, and seems to suggest the existence of a class of people who are extremely susceptible to its ill effects and who need not necessarily show any lesions of the skin. This is important, for bottles of this dye are issued in boxes containing notices warning people to test its effect on a small area of skin before using it, and are advised not to use it if a reddening of the skin results some hours after the experiment is made.

The action of the dye as a systematic poison may be delayed, for in the above case a day and a half intervened between the last exposure and the onset of symptoms ; as it was due to this delayed action that the patient had never associated his symptoms with his occupation, the question arises whether others may not be upsetting their health to a greater or less extent by dyeing their hair.

It would appear from the literature[1] on the subject of fur dermatitis that the toxic phenylenediamine becomes non-toxic when oxidised by hydrogen peroxide ; Bandrowski's base, which is formed, is an insoluble colouring matter which can be eaten by experimental animals without ill effects, but apparently the phenylenediamine is not always completely oxidised, and to prevent the furs remaining toxic they are washed for from seven to eight hours ; it is only when the washing has been scamped that the furs are toxic and cause dermatitis.

A similar process is used in hair dyeing ; the hydrogen peroxide and the phenylenediamine are mixed, and the resulting solution is applied to the hair, but unfortunately, a temptation exists not to wash too thoroughly, as the washing may spoil the colour. Worse still, some who dye themselves apply the solutions separately, by which means they obtain a finer and more lasting colour, but attended probably by greater risk of a toxic result.

If careless men dye heavy moustaches in this manner, and women with similarly dyed hair hold tresses of it between their lips whilst dressing, the possibility of poisoning is increased. So long, therefore, as people are willing to use this dye in quantity, elderly-looking people with suspiciously dark brown hair may occasionally be met with whose paroxysmal tachycardia or indigestion, whose palpitation or liverishness,

[1] *B.M.J.*, Epitome, January 19th, 1924.

or whose symptoms of gastric influenza will disappear if they will stop dyeing their hair.

But perhaps the most serious aspect of this case is the medico-legal one, for it cannot be doubted that Nott's patient was not far from death on the night he fell unconscious, and if he had been found dead, and an inquest had been held, would the true cause of death have been ascertained ?

Analysis. The phenylenediamines are readily soluble in ether, and may be extracted by that solvent from a solution made alkaline with sodium hydroxide.

Meta-phenylenediamine, dissolved in dilute sulphuric acid and treated with sodium nitrite gives a colour varying from yellow to red or brown (Bismark Brown).

With diazo-benzene (prepared by the action of sodium nitrite on a cooled solution of aniline in dilute sulphuric acid), *m*-phenylenediamine gives a red colour.

Para-phenylenediamine, warmed with a solution of ferric chloride in presence of hydrogen sulphide, gives a blue colour ; mixed with phenol and oxidised (*e.g.*, by boiling with manganese dioxide and sulphuric acid) it gives a dark blue colour ; oxidised at ordinary temperatures (*e.g.*, by hydrogen peroxide) in presence of aniline, it gives a blue colour.

Poisoning by Phenylhydroxylamine

The following case was described by Drs. Hirsch and Edel, physicians to the Municipal Hospital of Charlottenburg, Berlin. A student of the technical academy, while working in the chemical laboratory, broke a bottle containing an alcoholic solution of phenylhydroxylamine (C_6H_5NHOH), which saturated his clothes and flowed over his abdomen and the inner side of his thigh. He felt at once an intense burning of the skin, and retired to the lavatory to apply a cold-water bandage. About fifteen minutes afterwards he was found there in an almost helpless condition, being comatose and pulseless, with stertorous respiration and no reflexes of the cornea or pupils. When he was brought to the hospital the physicians believed him at first to be moribund. The alteration in the colour of the skin was very singular. The lips and mucous membrane of the mouth were greyish-blue, and the skin of the extremities was an intense blue colour, which contrasted peculiarly with the cadaverous aspect of the other regions. There were also numerous reddish-brown spots on the hands, thighs and abdomen, which did not disappear on pressure with the finger. Respiration was deep and regular, but the cardiac action was extremely weak. Injections of camphor, a hot bath, and other excitants having no visible effect, venesection was performed, and 300 c.c. of dark-brown, chocolate-like blood were drawn off. An intravenous injection of one litre of a solution containing 0·3 per cent. chloride of potassium and 0·4 per cent. bicarbonate of potassium was given immediately afterwards, whereupon the pulse and respiration rapidly improved and the patient in course of time regained consciousness. He vomited a large quantity of brown-coloured mucous fluid. During the first hours no urine was passed, and the catheter had to be used ; the urine thus evacuated contained much albumen and a rather large quantity of casts. During the first day the skin retained its peculiar colour. The patient passed a comfortable night, and on the following day, though still very weak, he felt otherwise well. The pulse became better, the urine was voided spontaneously, and the albumen and casts disappeared on the following day. The lips were now of a pale rose colour, the skin was very pale, and on the third day the normal aspect was restored. The patient soon recovered and was able to leave the hospital. Microscopical examination of the blood proved that the form of the red blood corpuscles was not changed, but that the haemoglobin had almost completely left them, and the spectroscope showed that the haemoglobin was altered into methaemoglobin, to which was due the brown colour alike of the blood, the vomited matter, and the little spots on the

skin. The case is very like one of poisoning by nitrobenzol, from which phenylhydroxylamine is derived. The alterations of the colour are characteristic of both drugs, but nitrobenzol never acts as quickly as the other. In the cases of nitrobenzol poisoning hitherto described, the full toxic action did not appear before one or two hours ; but in the present case the patient was in a helpless state in fifteen minutes, the poison having been absorbed by the skin only and not by the stomach. The characteristic smell of oil of bitter almonds was also absent. Phenylhydroxylamine is, therefore, a violent poison, which rapidly acts on the blood, the heart, and the kidneys.[1]

REFERENCES

Hirsch and Edel, *Berl. klin. Woch.*, 1895, 32, 891, 915.
Lewin, *Arch. f. Exp. Path.*, 1894–5, 35, 401.
Lewin, *Deutsche med. Woch.*, 1906, 32, 710.

Poisoning by Dinitro-phenol and Dinitro-cresol

Dinitro-phenol caused numerous cases of poisoning among French munition workers during the war of 1914–18, when it was used as an intermediate in the manufacture of explosives. The symptoms consisted of headache, dizziness, vomiting, profuse sweating, and high fever. A number of deaths occurred. The sweating and hyperthermia are apparently due to the fact that dinitro-phenol markedly increases the metabolic rate. It was at one time[2] recommended in the treatment of obesity, and although all investigators agree that its use should be carefully controlled by physicians, it was marketed in America as "Nox-Ben-ol." Dinitro-cresol, with similar therapeutic and toxic effects, but more active (so that a smaller dose suffices) has been studied in this country[3] and sold here under the name "Dekrysil." Although their use has been abandoned by physicians these substances still appear in nostrums and isolated cases of poisoning still occur.

The recommended therapeutic dose was 1–5 mgrm. per kilo bodyweight in the case of dinitro-phenol, and 1–2 mgrm. per kilo body-weight in the case of dinitro-cresol, the drug, in both cases, being given by mouth. These amounts were given daily for some weeks. In various animals the toxic dose is 10 mgrm. per kilo bodyweight. Koelsch[4] reported tolerance of doses of 20–30 mgrm. per kilo, but death after 50 mgrm. per kilo. Chronic rheumatism, alcoholism, tuberculosis, and liver or kidney inefficiency cause lessened resistance to the drug.[5]

Even with therapeutic doses of the drug, unpleasant symptoms have been observed in a number of cases. Among 113 obese persons treated with dinitro-phenol,[2] sixteen suffered from unpleasant reactions—cystitis in one case, derangement of the taste sense (inability to distinguish between sweet and salt tastes) in six, gastro-enteritis (possibly merely intercurrent) in one, and dermatitis in eight. The skin rash was urticarial and accompanied by intense itching. It disappeared, with soothing treatment, in two to five days, after the drug was stopped. Some patients became slightly jaundiced in appearance, and the serum was yellow. This, however, was probably due directly to the drug (which is yellow in neutral or alkaline solution) and if its colour was discharged by addition of HCl, the serum gave normal values for the icteric index.

[1] *Lancet*, 1895, 2, 1261.
[2] Cutting *et al.*, *J. Amer. Med. Assoc.*, 1933, 101, 193, 1472, 2099.
[3] Dodds and Robertson, *Lancet*, 1933, 125, 1137, 1197.
[4] *Zenitr. Gewebehyg.*, 1927, 4, 261 ; *Chem. Abs.*, 1928, 22, 4656.
[5] Perkins, *Pub. Health. Rep.*, 1919, 34, 2335.

Anderson, Reed, and Emerson[1] report the following case of poisoning by dinitro-phenol in therapeutic doses (thirteen other patients received the drug without untoward effects). A death from overdosage was reported in the San Francisco daily press on August 28th, 1933, and another fatal case was reported by Tainter and Wood.[2]

The patient, a woman aged 43, white, married, complained of overweight, an increase in the past year of 45 kgrm.; present weight, 79·5 kgrm.; height, 171·4 cm. At midnight of the day following fourteen days of *a*-dinithro-phenol therapy (0·075 grams of the sodium salt, three times a day by mouth) the patient complained of severe pruritus confined to both elbows, a small area in the centre of her back, the cervical region just below her ears, and over both knees. At 6 a.m. the following morning a maculopapular erythematous eruption appeared in the regions noted with a slight swelling of the soft tissues. She was given, orally, four times a day for four days a powder containing calcium gluconate, 1 gram, and ephedrine sulphate, 0·035 gramme. Calamine lotion was applied to the affected areas. During the day the pruritus, eruption, and œdema became more severe and more extensive. Lassar's paste without acid was applied locally, and codeine phosphate was given by mouth in 0·033 gramme doses. Twenty-four hours after the onset the whole body, except face and scalp, was involved, with œdema of both arms, left shoulder, neck, and lobes of both ears, and erythematous lesions were found on the back, chest and abdomen, and all four limbs, especially over the joints. There was no nausea, diarrhœa, dyspnœa, dysuria, frequency, or discoloured urine. The skin manifestations were most acute from forty-eight to seventy-two hours after the onset, when, in addition to the pruritus, pain developed on movement of the fingers and all large joints, as well as in the palms of the hands and soles of the feet. On the fourth and fifth days the skin lesions, œdema, pruritus and pains subsided, and the only symptoms complained of were stiffness on movement of the large joints. Since then the patient has had severe pain in one or more of the joints without swelling of the soft tissues. Temperature, pulse, and respiration rate were normal throughout. The patient had previously suffered from chronic hypertrophic arthritis.

A fatality due to the use of dekrysil for "slimming" occurred in London and was reported in the daily press (April 1934).

Analysis. In fatal cases dinitro-phenol may be recovered from blood and viscera, whence, in the Stas-Otto process, it is extracted by ether from the acid solution. It is a yellow crystalline solid, melting at 114° C. The sodium salt melts at 297° C. The yellow colour is almost discharged by hydrochloric acid.

Derrien's test for urine containing end-products of dinitro-phenol metabolism. To 10 c.c. of urine add 1 c.c. of 10 per cent. sulphuric acid and 1 c.c. of 0·5 per cent. sodium nitrite. Shake, and keep in the dark for five minutes. In another tube prepare 2 c.c. of a 0·5 per cent. solution of beta-naphthol in dilute ammonia, add the urine, shake, allow to stand at least a minute, and add 10 c.c. of sulphuric ether. Shake, and allow the liquid to separate into two layers. The presence of dinitro-phenol end-products is indicated by a coloration of the ethereal layer ranging from violet to wine-red. A yellow colour is to be ignored.

Poisoning by Trinitro-toluene (T.N.T.)

Source and Method of Occurrence. Trinitro-toluene, $C_6H_2(NO_2)_3CH_3$, is a high explosive obtained by nitrating toluene. At ordinary temperatures it is a yellow solid and melts at about 80° C. When mixed with 40 to 60 per cent. ammonium nitrate it is known as "amatol."

[1] *J. Amer. Med. Assoc.*, 1933, 100, 1053.
[2] *J. Amer. Med. Assoc.* 1934, *102*, 1147.

and with 20 per cent. ammonium nitrate as "ammonal." Until 1914 little was known of the poisonous effects of this substance. Since that time, however, its manufacture as an explosive has led to careful observations, from which it appears that it caused a considerable amount of poisoning in munition workers.

Mortality and Fatal Dose. Nothing is known of the fatal dose, but the mortality among recognised and notified cases is about 33 per cent. About 83 per cent. of the cases developed after working with the substance for five to sixteen weeks.

Symptoms. The effects of T.N.T. poisoning can be grouped under several heads, *e.g.*, dermatitis, digestive troubles, blood changes and toxic jaundice with hepatic changes. Localised rashes, especially about the hands, wrists and face, are common, and are usually of an erythematous character. Pruritus is intense, and the rash ends with desquamation. Evidence of absorption of the poison is shown in a pallor of the face and an ashen-grey colour of the lips. Jaundice often occurs quite suddenly and may be associated with rapid diminution of the liver, dullness, albuminuria, coma and death in about three weeks from the onset of the symptoms.

Treatment. Prevention and treatment are based on the fact that the main channel of absorption has been shown by Moore to be through the skin, and include the control of the workers, adequate mechanical protection of the exposed parts of the body and efficient cleansing of the skin. The prognosis of the jaundice is grave.

Post-mortem Appearances. The chief *post-mortem* lesion is a great destruction of liver substance which may be reduced to half its usual amount. The outer surface is smooth, reddish in colour, with slightly elevated yellow areas. On section the normal lobule pattern is obliterated. Microscopically a large part of the liver tissue is found to have undergone complete destruction, accompanied by a proliferation of fibrous tissue. The kidneys show cloudy swelling and fatty changes in the tubules. Certain persons seem more predisposed to poisoning than others.

Webster's Test for Trinitro-toluene in Urine. Although T.N.T. and also the modifications formed from it in the body are easily soluble in ether, yet, if the urine containing the excreted compound be shaken up with ether, and the ether be separated and tested with alkaline alcohol, no coloration results, showing that the ether has failed to remove it. But if the urine be mixed with an equal volume of 20 per cent. sulphuric acid solution,[1] and again shaken out with ether, the ether separated and washed free of acid with water, and then tested with alcohol made alkaline with potash the pink colour characteristic both of T.N.T. and a reduction derivative of it formed in the body appears.

When the amount of T.N.T. in a sample is large, giving rise to a suspicion of contamination of the urine by contact with skin, hair or clothing, the urine should be extracted first with ether without adding any acid; if a pink colour then appears on adding alkaline alcohol, it is due to T.N.T. of *contamination*: all that is necessary now, to disclose

[1] By volume.

the modified T.N.T. which has passed through the system, is to acidify as directed above, shake out with ether again, separate the ether and test with alcoholic potash.

The colour obtained when T.N.T. urine is treated as in Webster's test is not due to T.N.T. itself, but to a substance (or substances) formed from it by the reduction of one of the nitro-groups. This substance is 2 : 6 dinitro-4. hydroxylamino-toluene. It is excreted in the urine, conjugated with glycuronic acid, from which it is liberated by the treatment with acid.

Test for Trinitro-toluene in Air. Kay [1] has described a very sensitive test, in which T.N.T. is absorbed by drawing a measured volume of air through 150 ml. of acetone, and, after concentration by evaporation at a temperature well below 82° C., an aliquot sample of the solution is treated with one-tenth of its volume of aqueous 20 per cent. (w/w) sodium hydroxide. Trinitro-toluene gives a red colour, developing within ten minutes at room temperature and remaining stable for thirty minutes. Quantitative estimation is made by comparison with standard acetone solutions of trinitro-toluene similarly treated, the best range being from 0·0004 to 0·004 mg. per ml. of solution.

See also "Trinitro-toluene Poisoning" (officially communicated by the Ministry of Munitions), *Lancet*, 1916, 2, 1026, and Special Report. No. 11, Med. Research Committee, 1918.

Sub-Group 6.—Poisoning by Petroleum or Paraffin

Source and Method of Occurrence. Under the term petroleum, or rock oil, are included various oils used for illuminating purposes, of peculiar odour, which spring from the ground in various parts of the globe, and consist of mixtures of various hydrocarbons and inflammable products.

American petroleum consists mainly of aliphatic hydrocarbons (paraffins); that from Eastern Asia is made up chiefly of aromatic hydrocarbons; while that from Russia, Galicia, etc., is a mixture of the two kinds. The crude oil is subjected to fractional distillation and split up into fractions (1) low boiling oil (boiling point up to 150° C.) known as raw benzine or naphtha; (2) lighting oil, variously known as kerosene, coal oil, paraffin oil, lamp oil, etc. (boiling point 150° to 300° C.); and (3) a residuum with boiling point above 300° C. In America this residuum yields lubricating oils, semi-solid paraffin (vaseline) and paraffin wax. The low-boiling "benzine" fractions are further separated into various fractions, *e.g.* (1) petroleum ether or rhigoline, boiling between 40° C. and 60° or 70° C.; (2) petrol of various grades, boiling between 70° C. and about 120° C., and used as motor spirit, as a cleaning agent (sometimes under the name of benzine or purified benzine), and as a solvent; (it consists primarily of pentane, hexane and heptane. Aero spirit contains varying quantities of octane); (3) ligroine (120° to 135° C.), used as an illuminant; (4) residual oil (above 135° C.), which is used for dissolving lubricant oil, lacquers, varnishes, oil colours, etc. Terapin, a solvent oil, containing a considerable amount of aromatic hydrocarbons, including naphthalene, is obtained by distillation of oriental petroleum. In its poisonous effects it resembles benzene rather than petrol. The

[1] *Canad. J. Res.*, 1941, *19* [B], 86, 89.

production of these various substances is one of the world's great industries to-day, and the majority of the cases of poisoning occur in connection with the recovery of the petroleum from the wells, the storage in the steamers, the refining processes, and from the inhalation of petrol fumes in various processes connected with internal combustion engines, in garages, in rubber factories, cleaning establishments, etc. Apart from general intoxication, numerous skin affections may occur in the form of eczema, papillomata, etc.

Toxicity and Fatal Dose. In general the poisonous properties of petroleum cannot be rated very high; but many varieties of crude petroleum are much more poisonous than others and than the residual petroleums. In adults a wine glassful may not kill, and in one case five fluid ounces did not cause death. Even in the case of children the fatal dose is a very large one; and in the event of death supervening, it is in most cases due rather to the secondary result of local action of the poison on the stomach and intestines than to the result of the absorption of hydrocarbons. In children, 10–15 grammes of petrol are said to have caused death; an adult died after 30 grammes, but another recovered from 250 c.c.

Cases of poisoning from the inhalation of petrol fumes are common in industry, since if the concentration is high, mild intoxication will mean low concentration. One part per 1,000 is said to produce drowsiness in 15 minutes and vertigo and ataxia in an hour, and 7 parts per 1,000 intoxication in five minutes. In dogs 10 parts per 1,000 caused convulsions, 25 parts per 1,000 caused death.[1 2 3]

Mild symptoms are similar to those of alcohol or ether, there may be excitement, irritability and loss of control. With higher concentrations there may be drowsiness, ataxia, headache and vertigo. The vision may be blurred and there may be contractions and notching of the muscles.[3]

With still higher concentrations there may be disorientation, hallucinations, severe disorders of conduct, convulsions and coma. Serious headache is common.

The patient is cyanosed, the breathing stertorous and the skin cold and clammy. The reflexes may be diminished or absent in coma but may be exaggerated in convulsive states; there may be clonus and Kernigs sign may be present. The pupils are dilated and unequal. The urine may contain traces of albumin and sugar.

Post-mortem. The smell of petrol may be obvious when the body is opened. The lungs are œdematous; petechial hæmorrhages and larger hæmorrhages into the mucous membranes and subserous tissues may be found in the trachea gastrointestinal tract, bladder and brain. Cloudy swelling and fatty degeneration of the liver and of the proximal convoluted tubules of the kidney may be observed. Degeneration and chromatolysis of the nerve cells of the brain and spinal cord are described.[2]

Sequelæ, headache, sleeplessness and anorexia are common, but there may be less frequently definite organic change in the central nervous system. Symptoms may occur several months after the accident.

[1] Haggard, H. W., *Jour. Pharmacal.*, 16, 401 (1921).
[2] Machle, W., *J. Amer. Med. Ass.*, 117 : 1965 (1941).
[3] Rudd, T. N., *Jour. R.A.M.C.*, 82 : 271 (1944).

Treatment. If the petrol has been ingested immediate gastric lavage is essential. In inhalation cases artificial respiration with inhalation of oxygen combined with 5 per cent. carbon dioxide is usually necessary.

If the clothes are splashed they must be at once removed and the skin washed.

Sedatives may be required and the patient must be kept under careful supervision for several days in view of the liability to pulmonary complications and circulatory failures.

Analysis. The detection of petroleum, either in the vomited matters or in the contents of the stomach, is effected by distilling the suspected matters and determining the physical and chemical properties of the distillate. Since, even with petrol, some of the hydrocarbons boil at 120° C., and in the case of kerosene the boiling range extends to 300° C., an oil bath is desirable, and a water-cooled condenser should be used only during the earlier stages of the distillation. The notable features are the peculiar unpleasant odour and the inflammability of the distillate. Petroleum is insoluble in water, but is soluble in absolute alcohol, glycerine, ether, and ethereal and fatty oils.

Case. A child, aged fourteen months, whilst playing, swallowed some paraffin oil. She coughed and became unconscious. Four fits, each lasting about three minutes occurred. They were characterised by rigidity of the limbs, turning up of the eyes, and blueness of the face. Between the fits the rigidity passed off, but the unconsciousness remained. There was no vomiting. When admitted to the Manchester Infirmary the child was much collapsed and was unconscious; the respirations were slow (ten), deep, and sighing; the pulse was fairly good and not much increased in frequency; there was no cyanosis; the pupils were medium sized and equal; there was an odour of paraffin oil from the mouth. An attempt to wash out the stomach failed, as the eye of the catheter repeatedly became blocked with mucus. Respiration then ceased, and cyanosis occurred. Artificial respiration was carried out, and the cyanosis passed off, leaving the child very pale. The pulse ceased, death taking place one hour and fifty minutes after the swallowing of the paraffin oil. The amount swallowed is stated to have been about an ounce and a quarter. At the necropsy the lungs had the odour of paraffin oil, the œsophagus was slightly congested, and the stomach was very pale and contained much stringy mucus and globules of paraffin oil. The noteworthy points in this case are the convulsions and the absence of the usual irritant symptoms. The former are to be explained, no doubt, by the proclivity to convulsions which exists in infancy.[1]

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Poisoning by Pyridine (C_5H_5N)

Source and Method of Occurrence. Pyridine is a base forming salts with acids, obtained from bone oil and many organic substances by dry distillation. It is one of the nuclei of nicotine and is contained in the fumes of tobacco smoke. Pyridine is a strongly alkaline, irritant poison, as well as a drug possessing a profoundly deleterious effect on the medulla, and especially on the respiratory and cardiac centres situated therein. It diminishes reflex irritability, producing slowing of respiration and fall of blood-pressure, with nausea, diarrhœa, and profuse sweating. The drug rapidly appears in the breath and the urine.

[1] *Lancet*, 1, 1013 (1898).

A fatal case of poisoning is reported.

On August 29th, at 10.30 p.m., T. N., aged twenty-nine, a strong muscular man, employed as a stillman at some tar works, was admitted into hospital. He stated in thick guttural speech that he had swallowed " half a cupful " of pyridine bases at 3 p.m. on the same day, and during the intervening seven hours had been at home and vomited five times, the vomited matter having the smell of pyridine.

On admission he was pale, with slightly cyanosed lips and a dry white tongue, and was perspiring freely. The temperature was 103·4°, the pulse 128, weak and intermittent, respiration 40. The breathing was noisy, due to coarse mucous râles.

He complained of tightness in breathing, a choking sensation, pain down the centre of the chest, and pain over the stomach. Demulcents were ordered and given in ℥j doses, owing to the patient being unable to swallow more ; mustard and linseed poultices were applied to the throat and front of the chest, and an enema of ℥iij of brandy given and retained, after which the pulse and breathing improved, and in an hour he could swallow ℥ss of milk with ℥j of brandy. During this time the expectoration was white and frothy and had the odour of pyridine, as also had his breath.

On August 30th, at 12.30 a.m., the temperature was 104°, but gradually subsided to 99° at 9 a.m. During the night the bowels acted six times, the fæces being partially formed and of a light yellow colour, with no odour of pyridine. ℥x of medium straw-coloured urine were passed, with a specific gravity of 1020, no abnormal constituents being detected. At 8 a.m. the expectoration began to be purulent, the odour of pyridine having disappeared. On examination the lungs showed signs of acute congestion and bronchitis, but the patient felt much more comfortable. About 4 p.m. he began to be delirious, the temperature having risen to 104°. He continued wildly delirious, with a temperature varying from 104° to 105·8° all night, until 8 a.m. on August 31st, when he became quieter, and died at 10.15 a.m. forty-three hours after the accident. During the twenty-four hours ℥ix of urine were passed and the bowels acted six times, the motions having similar characters to those of the previous day.

At the *post-mortem* examination, made twenty-nine hours after death, the epiglottis was found congested on its under-surface. The larynx and trachea were lined by a friable yellow membrane, the large bronchi contained purulent matter, and were lined by a similar membrane. The lungs were congested and œdematous. Nothing was observed about the mouth, tongue, or fauces, but the œsophagus and cardiac end of the stomach were greatly congested, the pyloric end and the commencement of the duodenum being slightly congested. The only changes found throughout the intestines were a few small petechiæ in the small intestine. The liver was of normal size, and showed a few small fatty patches on its upper surface. No changes were detected in the heart, kidneys, or spleen, and no odour of pyridine was observed throughout the examination.

At the inquest it transpired that the man had filled a cask too full of commercial pyridine, and to reduce the amount, he obtained a bent piece of iron tube about three feet in length and one inch bore, one end of which he inserted into the cask, and to the other end, in order to start the syphon action, he applied his mouth and unfortunately drew in the fluid.

REFERENCE

Helme, " A Fatal Case of Pyridine Poisoning." *B.M.J.*, 2 : 844 (1893).

Group 7.—POISONS OF VEGETABLE ORIGIN

Of all the groups of poisons this is the most artificial from the point of view of action, for it includes illustrations of almost every action of which poisons are capable. Opium acting on the brain, strychnine on the cord, ergot on the vessels, coniine on peripheral nerves, primula on the skin, are illustrations that occur to the mind on a most cursory survey. However, the grouping has some convenience for reference, and is here adopted in deference to this convenience.

Poisoning by Abrus Precatorius (Jequirity—Abrin)

Jequirity is the seed of the Abrus precatorius. It is the size of a small pea of a beautiful red colour with a black spot on one pole. Jequirity became known in medicine after its introduction in 1882 by de Wecker, who used the infusion for producing conjunctivitis in connection with the treatment of trachomatous pannus. It produces a violent reaction unless care is used. In 1891 Hellin separated out the poisonous principle *abrin*, an antigenic substance which, by inoculation, gives rise to a true specific anti-abrin. It is an exceedingly violent poison which produces symptoms similar to those of Ricin (which see.) The preparation of Merck's used by Ehrlich, killed mice in a dose of 1 : 100,000, and even 1 : 800,000 produced great loss of hair. Very dilute solutions produced marked conjunctivitis both in animals and man. In at least one case an experimenter suffered severely in this respect. The lesions in guinea-pigs are practically identical with those produced by ricin with the exception of the fact that abrin produces a severe local reaction.

The powdered seeds are commonly used in India for killing cattle and sometimes for homicide. On injection under the skin death is produced within about 24 hours with symptoms similar to those of snake poisoning.

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Poisoning by Aconitum Napellus (and other species)

Source and Method of Occurrence. Aconitum napellus is not a native of Britain, but is often grown in gardens for its showy flowers. There are several other species of aconite, all containing different aconitines, which, however, are closely related.[1] Japaconitine appears to be more poisonous than aconitine, and pseudaconitine (from *A. ferox* - *A. Balfourii*) appears to be the most poisonous of all.

All parts of the common aconite (*Aconitum napellus*) are highly poisonous. The plant is commonly known by the names of monkshood, wolfsbane, or blue rocket.

In most cases it is the tincture, liniment, or other preparation which is taken in poisonous dose by misadventure.[2] Still more rarely the active alkaloid, *aconitine*, has been administered. This is one of the most deadly known poisons, one-sixteenth of a grain having proved fatal, and one-fiftieth of a grain is believed by some to be a fatal dose. The roots, seeds, and leaves of the plant, when masticated, produce a tingling followed by a numb sensation, affecting the lips, tongue and interior of the mouth generally. At first the root appears to be almost tasteless, as the effects are only manifested after the lapse of a brief interval. The sensation, once produced, lasts for several hours.

[1] See Evers, " Chemistry of Drugs," 1926, p. 115, and Henry, " Plant Alkaloids."

[2] The liniment is now the only official preparation.

The roots of *Aconitum ferox* (*A. deinorrhizum*), the Indian bikh or bisch, and those of Japanese aconite ("Bushi") from *A. Fischeri*, are also articles of commerce, and are more poisonous than the ordinary *A. napellus*. Other species of aconite, however, contain non-poisonous alkaloids.

The root has been eaten in mistake for horseradish, to which it bears a remote resemblance. It has also been administered for homicidal purposes.

A mistake of this kind led to fatal results in three hours in a case which occurred at Lambeth; and another set of cases occurred at Dingwall in 1856. Here three persons were poisoned by reason of their having had sauce, made with the root of aconite, served at dinner with roast beef in the place of horseradish sauce. They were healthy adults; and all died within three and a half hours.

Such mistakes show deplorable ignorance, but there is always the risk of their occurrence when horseradish and aconite are grown near to each other in a garden, at that season of the year when the leaves have fallen. A trial for murder by poisoning with the root of this plant took place at the Monaghan Lent Assizes in 1841.[1]

The deceased had eaten for his dinner some greens dressed for him by the prisoner; he complained of their having a sharp taste, and this was perceived also by another person present who tasted them. It was ascertained that soon after the meal the deceased had vomited some greenish matter, and suffered from purging, restlessness, incoherence, lock-jaw, and clenching of the hands. He died in about three hours after having eaten the greens, but was not seen by a medical man while living. The accused was convicted of murder, and confessed before her execution that the powdered *root* of aconite had been mixed with pepper and sprinkled over the greens.[2]

The use of aconite root for homicidal purposes though rare in Europe is common in the East.

A liquid sold for external use under the name of **neuraline** appears to be a preparation of tincture of aconite mixed with chloroform and rose-water. **Nervine** is also a preparation of aconite.

The case of Lamson, C. C. C., March 1882, is the only known case of the homicidal use of the alkaloid in England.

Alarming results have been known to ensue after the administration of pills, each containing $_2\frac{1}{5}_0$th of a grain of aconitine, four times a day. The symptoms were developed on the second day.[3]

Toxicity and Fatal Dose. The poisonous effects of all preparations are due to the alkaloid, aconitine. Aconitine decomposes readily, and therefore commercial preparations vary widely in strength, some being at least a hundred times more active than others. The medicinal dose BPC is 1/640 grain, and it appears to be dangerous in doses as low as $\frac{1}{5_0}$th grain; death has occurred from $\frac{1}{1_6}$th grain.

Of the *root* sixty grains have proved fatal; but it is probable that this is much in excess of the minimum fatal dose. Of the old (unofficial) pharmacopœial tincture two or three drachms might probably be fatal. Of *Fleming's tincture* twenty-five minims have killed an adult. Four grains of *alcoholic extract* have proved fatal. Of the old (unofficial, non-alcoholic) *extract* two grains have proved fatal. It is very uncertain in its action: but is much less active than the alcoholic extract. The

[1] *R.* v. *McConkey.*
[2] *Dub. Med. Jour.*, vol. 19, p. 403.
[3] *Lancet*, 1880, 2, p. 46.

liniment is stronger than even Fleming's tincture, and twenty minims would probably form a fatal dose.

The tincture of the root operates powerfully in small doses. Dr. Male died from the effects of not more than *eighty drops* taken in ten doses over a period of four days, the largest quantity taken at once being *ten* drops.[1] Pereira had known tingling and general numbness of the limbs produced in hysterical women by a dose of only *five minims* of a carefully prepared tincture. Topham has published an account of the symptoms produced by *fifteen minims* of the tincture of the root of aconite.

Immediately after taking the poison in a mixture into which it was put by mistake, the patient (a woman, *æt.* 27) felt a sensation of numbness in the tongue, with difficulty of swallowing. There were convulsive twitchings of the muscles of the face, and she lost the power of walking. There was complete unconsciousnoss, which continued for two hours, when she began to recover. The pupils were observed to be slightly contracted. The intensity of the symptoms varied at intervals and came on in paroxysms. They indicated great disorder of the nervous system. The next day she had numbness in both arms, but she recovered rapidly and perfectly.

Duration. The symptoms appear rapidly and death usually occurs in two to six hours. With very large doses death may occur almost instantly.

Symptoms. Aconite acts powerfully upon nerve terminals and peripheral nerves, and death takes place from its effect upon the cardiac and respiratory centres.

The symptoms met with seem to be practically uniform. In from three to five minutes after chewing aconite root, or after contact of any of its preparations with the tongue, a hot, tingling sensation followed by numbness is experienced on the tongue, extending to the fauces and to the lips, especially the lower. The sensation soon becomes very severe, and is accompanied by salivation, and a sensation of swelling of the fauces, and there may be difficulty in swallowing. Later the feeling is one as if the tongue had been seared with a hot iron, and there may be complete paralysis of that organ. Vomiting usually sets in in an hour or two at the latest, and is usually severe and spasmodic. The patient feels cold, especially in the extremities, and the skin is cold, clammy, and perspiring. There may be a feeling of tingling and numbness extending over the whole body, or a sensation of impending paralysis. The patient is restless, and there may be twitching of the muscles, and great muscular weakness is complained of. Convulsions are common. Respiration is slow and laboured, the pulse slow and feeble at first; later it becomes irregular and rapid. The pupil is at first contracted but dilates towards the end. Death takes place from respiratory paralysis before the heart goes into fibrillation as a rule, although the drug acts strongly on the heart.

The symptoms suffered by a man who had accidentally taken aconite root were very characteristic. In *two* minutes he felt a burning heat in the mouth, throat, gullet, and stomach; then a sensation of swelling in the face, with a general feeling of numbness and creeping of the skin. Restlessness, dimness of sight, and stupor almost amounting to insensibility, followed; and in about an hour after the meal he was found speechless, frothing at the nose and mouth, the hands and jaws clenched,

[1] *Prov. Med. and Surg. Jour.*, August 20th, 1845, p. 535.

appearing occasionally as if dead, and then again reviving. Vomiting, purging, tenderness at the pit of the stomach, cramps, tingling of the flesh, and a burning taste in the mouth followed. This man did not entirely recover until after the lapse of five weeks.

Treatment. Empty and wash out the stomach, leaving in a suspension of animal charcoal. Atropine has been found of great value in alleviating the symptoms and in improving the respiration. It also improves the irregularity of the heart and delays the terminal fibrillation. Digitalin injected hypodermically may also be of value in supporting the heart. Artificial respiration must be adopted and persisted in if the respiration fails. Inhalation of oxygen with 6 per cent. of carbon dioxide may be used to stimulate the respiratory centre.

Post-mortem Appearances. A slight brown staining of parts of the stomach has been observed ; otherwise, there is nothing to be expected or looked for except pieces of the plant.

Analysis. The botanical characters of the root and leaves, when any portions can be obtained, will enable a medical witness to identify this vegetable poison. The root has been frequently, and fatally, mistaken for horseradish, but there are these striking differences :—

1. Aconite-root is very short, conical, and tapers rapidly to a point. 2. It is externally of an earthy-brown colour—internally white, and of an earthy smell—the cut surface is rapidly reddened by exposure to air. It has numerous long thin fibres proceeding from it. 3. It has at first a bitter taste, but after a few minutes it produces a disagreeable sense of tingling and numbness on the lips and tongue. 1. Horseradish root is long, cylindrical or nearly so, and of the same thickness for many inches. 2. It is externally whitish-yellow, and has a pungent odour when scraped. 3. Its taste is sometimes bitter, but it produces an immediate hot or pungent sensation.

The leaves of aconite or monkshood are of a dark-green colour, thick, and of a peculiar shape. When masticated, the leaves slowly produce on the lips and tongue the persistent sensation of tingling and numbness, with the sense of coolness. They are less powerful than the root and seeds. The seeds differ in appearance from those of other poisonous plants. In any suspected case of poisoning by aconite, the vomited matters, or the stomach and intestines after death, should be carefully examined for portions of vegetable matter which may be compared with the structure of the undoubted aconite plant.

Aconitine may be extracted from organic liquids by the Stas-Otto process for the separation of the alkaloids. In this way and by applying the test of taste and that of physiological action on animals (mice or frogs), a very minute trace of aconitine may be detected. No other alkaloid produces the same persistent sensation upon the tongue as the alkaloid, or mixture of alkaloids, known as aconitine. (Veratrine produces a somewhat similar though much weaker effect.) Aconitine yields the general reactions of the alkaloids, and various colour tests have been described, but are of very doubtful value. The response to the general alkaloid reagents is as follows : Iodine solution gives a reddish-brown precipitate ; mercury potassium iodide a yellowish-white precipitate, gold chloride and picric acid yellow precipitates, phosphomolybdic and phosphotungstic acid white precipitates, while mercuric

chloride, platinic chloride, potassium ferrocyanide and ferricyanide give no precipitate except from very concentrated solutions. From a solution of aconitine, made faintly acid with acetic acid, potassium permanganate precipitates microscopic red crystals. These tests are all successful with a solution containing 0·25 mgrm. of aconitine per cubic centimetre, and some of them with still more dilute solutions.

Nevertheless, physiological tests are of more value than chemical in this case. The peculiar effect on the tongue, already described, is noticeable with a solution containing 1 mgrm. in 500 c.c. The poisonous effect upon mice or frogs may be compared with that of pure aconitine. Führer (*Arch. Exp. Path. Pharm.*, 1911, 66, 178) describes a test depending on the action of aconitine on the isolated heart of the frog. These effects consist of a preliminary slowing, sudden acceleration then inco-ordination. Extra ventricular systoles occur and the rate of beating of the ventricles increases above that of the auricles until there is complete inco-ordination with "peristalsis" of the heart muscles. Finally the irregularity passes off, the heart beats more regularly but weakly and slowly—the ventricle then stops followed by the auricle.

Aconitine readily decomposes when in alkaline solution; and hence it speedily disappears from liquids which remain alkaline, and can no longer be detected. Sir Thos. Stevenson found that its presence could no longer be detected in viscera where it was known to exist, should these become and remain alkaline for some time from putrefactive decomposition. The active alkaloids, aconitine and isaconitine, seem readily to undergo hydrolysis, especially in alkaline media. Thus aconitine in ammoniacal mixture speedily splits up into another base aconine, and benzoic acid. It follows that in the Stas-Otto extraction method care must be taken to prevent hydrolysis—*e.g.*, by avoiding alkalinity of the fluid as much as possible, and by keeping the temperature low.

In the Lamson case, from a portion of the first ejected vomit, from the urine drawn off from the bladder after death, and from the stomach, stomach contents, liver, spleen, and one kidney, taken together, Sir Thos. Stevenson and Dupré extracted aconitine by a modification of Stas's process. The existence of this was proved by its general reactions as an alkaloid, by the peculiar sensation which it excited upon the tongue, and by comparison of its fatal effects upon mice with those produced by Morson's aconitine. One two-thousandth part of a grain of English aconitine may be recognised by the taste-test, and the same quantity will kill a mouse within a few minutes.

Cases. A case of poisoning by German aconitine (Merck's) is thus recorded. An analytical chemist took eight grains of aconitine after dinner, with suicidal intent. Half an hour later the first violent symptoms appeared. A burning sensation in the mouth and throat first made itself felt, and this became more intense every minute; intense pains in the stomach ensued after thirty minutes, and these became so violent in a few seconds that the patient writhed, shrieking in the most dreadful convulsions, and trying to strike the wall with his head. Very soon he became incapable of swallowing; he was seized with spasmodic cough, and wanted to vomit. In spite of emetics, he could not vomit, however, till an hour after taking the poison, and then with great exertion a dark-greenish fluid was ejected; but this afforded no relief to the pain in the stomach, and the burning sensation in the throat, which rendered swallowing difficult. The application of the stomach-pump afforded no relief. Exhaustion ensued after violent convulsions, and the symptoms reappeared with renewed force. At the beginning

of the third hour, the pain and convulsions attained such violence that death was expected every instant. In the fourth hour, after repeated injections of morphine, the patient seemed somewhat better. Previous to this he indicated that his skin was greatly irritated. This irritation of the skin, as of ants crawling, continued apparently the whole time, and whenever the intensity of the pains somewhat remitted, he scratched the skin of the face and breast in a convulsive manner till these were sore. His eyes glared wildly, sometimes resting with a fixed stare on one point. The convulsions were repeated at almost regular intervals, and the inclination to vomit continued, although vomiting did not continue after the second hour. At intervals of about forty minutes the patient seemed to lose consciousness, but only for a few minutes, and then the convulsions and other symptoms reappeared with undiminished violence. Three hours after the onset of the symptoms he became incapable of intelligible utterance, but indicated that he felt giddiness; soon after he appeared to lose sight. He threw himself wildly about on the couch, screamed and uttered fearful groans. Exhaustion and apparent coma ensued, and then renewed attacks of the most violent description. Difficulty of breathing set in, and he appeared to suffocate. The pulse and body temperature fell considerably, and before death, which occurred at the end of twelve hours, exhaustion and unconsciousness set in, cold perspirations, and death-like pallor. Though death from asphyxia was all along expected, it occurred from syncope. The *post-mortem* appearances showed nothing unusual. The pupils were dilated, the interior of the mouth was pale, the brain and lungs were congested, heart normal, the liver and kidneys were congested. There was inflammation of the stomach, and its mucous membrane was congested (*sic*). The alkaloid was found by chemical analysis in the contents of the stomach; but, very remarkably, none was found in the urine of the deceased.[1]

On December 3rd, 1881, Lamson, a medical practitioner, visited his brother-in-law, *æt.* 19, who was at a school in Wimbledon. John, though a cripple, and paralysed below the pelvic region, was at that time in good health. In the presence of the master, Lamson gave to John a gelatine capsule, which he pretended to fill with powdered sugar, but into which he no doubt introduced a fatal dose of aconitone—perhaps the whole of two grains which he had purchased a few days previously. This was done under the pretence of showing the youth how to use the capsules for taking nauseous medicines. Lamson then left. Twenty minutes or half an hour afterwards the victim was seized with pain in the stomach, which he at first called heartburn, and which he compared to pain which he had experienced on a former occasion when Lamson had given what professed to be a quinine pill or powder. In a box belonging to John there was found, after his death, a packet of quinine powders, some of which were mixed with aconitine, whilst others were free from that poison; and also pills containing quinine and aconitone. There is no doubt that attempts had been made on John's life on two previous occasions by the administration of these articles furnished to his brother-in-law by Lamson. The boy was taken upstairs and he vomited, and was in great pain. He said his skin felt all drawn up, and that his throat burned. When first seen by Berry one hour and forty minutes after the administration of the poison, he was lying on the bed, with great pain in the stomach. He complained of the skin of his face being drawn, of a sense of constriction in the throat, and of being unable to swallow. He retched violently and vomited a small quantity of dark brown fluid. Half an hour later he was also seen by Little, and two hours and three-quarters after the poison was swallowed, a quarter of a grain of morphine was injected beneath the skin. This somewhat eased the patient's agony; but the symptoms returned with increased severity. At one time he was with difficulty kept lying down by the united force of two men. An hour later the morphine injection was repeated—one-sixth of a grain being used. Twenty minutes later he died, having been conscious almost to the last. Death occurred four hours and five minutes after the administration of the capsule, and not quite four hours after the commencement of symptoms. At the *post-mortem* examination, made by Bond, the only unusual appearances were—redness and inflammation of the cardiac end of the stomach, which had a blistered appearance; great congestion of the first portion of the small intestine (duodenum), and patches of congestion in other portions of the intestine in a lesser degree. The brain was hyperæmic. The membranes of the spinal cord were congested. The lungs were much congested, more especially towards

[1] *Med. Press*, May 24th, 1882, p. 439.

the posterior parts. The heart was very flaccid and as if sodden and stained with blood-pigment.[1]

The case of the man Hunt, who, in 1863, destroyed his wife and children by prussic acid, presents some features of interest in reference to the symptoms and appearances produced by tincture of aconite. The quantity of tincture taken by him was not determined; but the man was soon afterwards seized with violent spasmodic retching, the face was pale, the skin cold and clammy, the pulse small and hardly perceptible, and the action of the heart feeble. The pupils were much dilated and the eyes brilliant and sparkling, the breathing quiet and regular, except during the fits. He complained of pain in his heart. In attempting to walk, he staggered and had no power to raise his arms. He was perfectly conscious, called for writing materials, and wrote a few lines. He then became suddenly worse, and a quarter of an hour before his death he lost all power and sensation in his limbs, the sharpest pinches producing no impression. The pulse was imperceptible. There were no convulsions, but complete relaxation of the limbs at death, which appeared to arise from syncope three-quarters of an hour after he had taken the poison. On *inspection* forty-two hours after death, there was great rigidity of the muscles. The substance of the brain was firm and healthy; the vessels on the surface were filled with blood. The heart was healthy; the right side was greatly distended with dark fluid blood; the left side contracted and quite empty. The lungs were healthy. In the abdomen the viscera were healthy, with the exception of the stomach and duodenum. The mucous membrane of the stomach had a bright red colour at the larger end. There were marks of irritation, with softening and separation of the mucous lining, the whole of the membrane being in a highly corrugated condition. Traces of aconite were found in the contents of the stomach. The deceased had provided himself with an ounce of the tincture of aconite, and had swallowed the greater part of this mixed with water.

The following note on a case is taken from the *B.M.J.*, Epit., 1897 :—

Robinson[2] reports the case of a soldier who, after a debauch, took about two drachms of tincture of aconite. He was seen an hour later, when he was recumbent, tossing his limbs about and complaining of numbness and cramps in the arms and hands; his radial pulse was imperceptible, carotid 119, respirations 19, pupils slightly dilated but sensitive, nose pinched, extremities cold, face bedewed with cold sweat; at times he lapsed into unconsciousness. Between one-tenth and one-fifth grain of apomorphine hypodermically produced vomiting, and the stomach was thoroughly washed out by means of a tube. At intervals in the course of four hours—by which time he was out of danger—he was given hypodermic injections, amounting in all to twenty-five minims of tincture of digitalis, forty-five minims of aromatic spirits of ammonia, and two drachms of brandy. Robinson considers that digitalis is far superior to atropine or strychnine as an antidote to aconite, but that stimulants must also be used to gain time for the digitalis to act.

The following is one more illustration of the folly of keeping liniments, etc., in ordinary bottles alongside medicine for internal use :—

On December 12th Mr. T. T. T., aged seventy-five, about 10.50 p.m., went from his bedroom into an adjoining room with the intention of taking a bronchial mixture, but by mistake took hold of a three-ounce bottle containing equal parts of lin. aconiti, lin. belladon, and lin. chloroform, of which he swallowed twelve drachms before he discovered his mistake. A relative at once administered an emetic of mustard and water.

At 11 p.m. he was found sitting supported on the side of the bed, retching violently. Only slight emesis had occurred. His face, which had an expression of extreme anxiety, was covered with a clammy perspiration, and was drawn and pallid. The pulse was full and regular. He complained of a burning sensation in the epigastric region and extreme suffocation.

Apomorphine one-tenth grain injected hypodermically, followed by one ounce of brandy with water by the mouth, produced emesis in twenty seconds (very slight). As violent retching continued without further emesis, another hypodermic was given but failed to produce vomiting. His speech now became lost, and the arms and hands were in a state of clonic spasm. They rapidly became fixed tonically

[1] *B.M.J.*, 1913, 2, 1306.
[2] *Bost. Med. and Surg. Jour.*, August 25th.

in an arched position in front of the chest, with the hands midway between pronation and supination and the thumbs flexed into the palm. The legs were flexed on the thighs and the thighs on the abdomen.

A hypodermic injection of digitalin, one-fiftieth grain, was now given and a sinapism applied to the cardiac region, but nevertheless the pulse became feeble and irregular and the heart's action weak. He now became unconscious; the pupils were dilated and did not react to light, and there was no corneal reflex. The lips were blue and the face livid, with frothing at the mouth. At this time, twenty-five minutes after the swallowing of the mixture, the radial pulse became imperceptible and the heart's action very irregular. The body and extremities were cold and clammy, and the urine escaped involuntarily.

A hypodermic injection of twenty minims of ether was administered and artificial respiration commenced, but at 11.30 p.m. the patient expired.[1]

In 1880 three cases of poisoning by crystallised nitrate of aconitine occurred in Holland,[2] one of which proved fatal.

The first was the case of a weakly man, sixty-one years of age, suffering from chronic bronchitis, and a febrile attack. For this there was prescribed a solution of nitrate of aconitine. The patient took five drops, containing ·006 of a grain of the nitrate, at 7 p.m. This produced an astringent and burning taste in the mouth, extending to the stomach. At 9 p.m. the dose was increased to twenty drops (= ·025 of a grain); and this dose was repeated at 8 a.m., 11 a.m., 4 p.m., 9 p.m.; next day, at 10 p.m., a final dose of ten drops (= ·012 of a grain) was taken. In all one-seventh of a grain of the nitrate was taken in seven doses. After each dose the patient was seriously indisposed, so that eventually his life was in jeopardy. The symptoms were a feeling of coldness, cold, clammy perspiration, severe vomiting, difficult respiration, great lassitude, and the patient felt as if he were about to become paralysed. There were intermittent deafness and blindness and spasmodic twitchings of the whole body, but more especially of the muscles of the face. At one time he felt that he was dying, and stated that he had been poisoned. The respiration became stertorous and quickened; then slow and gasping. There was no loss of consciousness. It is not stated that there was any loss of sensation or any actual paralysis.

In the second case, a man, *æt.* 62, took an undetermined dose of the same medicine. When seen he had cold, clammy perspiration, a weak, irregular dicrotic pulse, and was conscious. The respirations were short, laboured, irregular and superficial. The pupils were contracted, and responded feebly to light. There was no difficulty in swallowing. There was great precordial anxiety, and *facies hippocratica*. Suddenly the pulse entirely ceased, though the cardiac beats could still be feebly heard; and a deathly pallor supervened. The patient rolled from side to side of the bed. The pupils were now dilated. Tonic convulsions of the facial muscles set in, with trismus; then, three hours after the dose, general clonic convulsion, and the patient lost consciousness. In five or six minutes muscular relaxation ensued, but the convulsions returned in a quarter of an hour. An hour later death appeared imminent. Vomiting now set in, the pulse improved, and in twenty-one hours the man was convalescent.

The third case terminated fatally. Dr. Mayer, who had prescribed for the above patients, himself took from fifty to sixty drops of the solution of nitrate of aconitine, prescribed for the first patient. This corresponds to one-thirteenth to one twenty-first of a grain of the nitrate. It may be assumed that the dose was probably one-sixteenth of a grain. The symptoms commenced in an hour and a half; but they were not accurately noted till 8 p.m., four hours after the alkaloid had been taken. He was then found with a small, weak, irregular, but not slow pulse, cold skin, and contracted pupils. He had an astringent and burning pain in the mouth, extending to the stomach, and difficulty in swallowing. The tongue was swollen. There was great precordial anxiety. He complained of burning pain, weakness, and heaviness of the limbs—especially the lower—which felt cold. Suddenly vision was lost, and the pupils became dilated. Soon, however, they again contracted, and vision was restored. Vomiting was procured by tickling the fauces. At 4.40 p.m. severe convulsions first set in, with stertorous respiration, singing in each ear alternately, and deafness. Ether was employed hypodermically, and

[1] *B.M.J.*, 1896, 1, 399.
[2] Schmidt's *Jahresb.*, 189, p. 122; *Berl. klin. Woch.*, 1880, p. 337.

its use was followed by renewed vomiting and convulsions. The pulse nevertheless improved, and ether was again injected. In a few minutes there was renewal of severe vomiting and convulsions, and the patient became unconscious; the pulse failed, and death ensued at 9 p.m., without return of consciousness, five hours after the administration of the fatal dose. On *post-mortem* examination the viscera were unusually charged with blood, and there was considerable hyperæmia of the stomach and small intestines, so that the colon and rectum appeared pale and bloodless by contrast. The intestines contained fæces, there having been no stool passed during the illness; and the bladder contained two and a half ounces of urine.

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Poisoning by Æthusa cynapium (Fool's Parsley) [Umbelliferæ]

Source and Method of Occurrence. Fool's parsley, or lesser hemlock, is common in gardens in some districts. The leaves so closely resemble those of parsley that they have often been gathered for them by mistake.

That the root of this plant contains a poison, and that it is capable of producing fatal effects is shown by a case in which death took place in an hour.

Leschke [1] says that fool's parsley contains coniine. It is also stated to contain an alkaloid cynapine which is also present in Æthusa fatua, but is apparently not dangerous to animals.[2]

The death of two Italian prisoners of war is reported from eating the root which they mistook for an edible Italian root.[3]

Two ladies partook of some salad, into which the leaves of this plant had been put by mistake for parsley. They soon experienced nausea, with occasional vomiting, oppressive headache, giddiness, and a strong propensity to sleep, though this was prevented by frequent startings and excessive agitation. There was a sensation of pungent heat in the mouth, throat and stomach, with difficulty of swallowing, thirst, and loss of appetite. There was numbness, with tremors of the limb. The two patients only slowly recovered from the effects of the poison.[4]

Analysis. It is distinguished from garden parsley by the smell of its leaves when rubbed, which is peculiar, disagreeable, and very different from that possessed by the leaves of parsley. The leaves of fool's parsley are finer, more acute, and of a darker green colour. Its flower-stem, which is striated, or slightly grooved, is easily known from all other umbelliferous plants by the beard, or three long sharply reflexed bracts of the partial involucrum under the flower. The flowers are white: those of the garden parsley are of a pale yellow colour.

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[1] "Clinical Toxicology," 1934, p. 222.
[2] Lander, *Vet. Toxicol.*, 1912.
[3] *Pharm. Jour.*, 152 : 41 (1944).
[4] Churchill's "Botany."

Poisoning by Aloes (Sp. Var.) and other Vegetable Purgatives

Source and Method of Occurrence. These substances, such as Julep Podaphyceum, colveynth, elaterium, scammony, etc., contain active principles usually resinous, which cause irritation of the intestinal tract, colic and diarrhœa and effusion of mucus into the gut. They cause congestion of the pelvic viscera and also is stated to have a specific effect on the uterus. Colocynth [1] has occasioned death in several instances: in one case a teaspoonful and a half of colocynth powder destroyed life: and one drachm of gamboge, a medicine much used by quacks, has proved fatal to a man.[2] Aloes and colocynth mixed are said to be the basis of the quack medicine sold under the name of Morison's Pills.

These have proved fatal in many instances from the exhaustion produced by excessive purging from the large quantity taken in frequently repeated doses. Our knowledge of the symptoms and appearances produced by these irritants is, indeed, chiefly derived from the cases which have proved fatal under this treatment. In the seventeenth volume of the *Medical Gazette* will be found four cases of this description. The most prominent symptom is excessive purging, with the discharge of large quantities of mucus; the individual becomes exhausted, and slowly sinks. In some instances the symptoms are those of inflammation and ulceration of the bowels. In 1836 a man was convicted of having caused the death of a person by the administration of these pills; in this instance the death of the deceased was clearly due to the medicine, and on inspection the stomach was found inflamed and ulcerated; the mucous membrane of the small intestines was inflamed and softened, and there was the appearance of effused lymph upon it.

Holloway's Pills are of a more innocent description; the principal ingredients in them is aloes. In all cases it must be remembered that these drastic purgatives may cause serious symptoms, or even death, when administered to infants, or to persons debilitated by age or disease; and it is not necessary that the dose should be very large in order that the fatal effects should follow. The question here will be, whether the medicine caused death, or whether it simply accelerated it, although in a legal view that which accelerates causes. Probably many deaths are accelerated by self-administered purgatives.

Hierapicra (*Holy Bitter*) is a popular aloetic compound, and one death is reported to have been produced by it in 1837–8. There is reason to believe that it is occasionally used for the purposes of procuring criminal abortion. A man was tried and convicted of this offence,[3] and the noxious properties of this compound then became a subject of inquiry. The dose, and the condition of the woman to whom it is administered, will of course affect the answer to this question. At the trial above mentioned it was probably considered to be a noxious substance within the meaning of the statute. The fact that, under the names of *Pulvis Aloes cum Canellâ*, it was formerly admitted into the British Pharmacopœias cannot justify the mischievous uses to which it may be put. *Hierapicra* is a snuff-coloured powder of an intensely bitter taste. It consists of four parts by weight of aloes, and one part by weight of powdered canella bark. The proper medicinal dose was formerly fixed at from five to fifteen grains. Its injurious effects on pregnant females are chiefly due to the aloes. This specially affects the rectum, and by contiguity, under violent irritation or purging, may affect the uterus. From the taste and colour which it imparts to liquids, it is not probable that it could be taken by a female unknowingly.

Death has been caused by aloes taken in nitric acid; but in this case the mineral acid was most probably the destructive agent. A singular case occurred in Germany, where a medico-legal question was raised

[1] See also p. 674.
[2] Traill's "Outlines," 150.
[3] Aylesbury Lent. Ass., 1857, *R.* v. *White*.

respecting the poisonous properties of aloes. A woman, *æt.* 43, not suffering from any apparent disease, swallowed two drachms of powdered aloes in coffee. Violent purging supervened, and she died on the following morning, twelve hours after having taken the medicine. On inspection the stomach was found partially, and the small intestines extensively, inflamed. There were no other particular appearances to account for death, and this was referred to the effect of the aloes.

For the detection of aloes, see Allen's "Commercial Organic Analysis," 5th Edition, Vol. VIII, p. 79 ff.

Arrow Poisons

These, perhaps, hardly come within the range of a work on medical jurisprudence, for happily, the use of poisoned arrows is confined to certain tropical native races. Stockman, of Glasgow, delivered an interesting address on these poisons in 1898, which is well worth the reader's attention.[1] (See also poisoning by Curare, p. 687).

He states that the bushmen in South-West Africa use local poisons, differing in different districts. He mentions, on the authority of Baines, a poison made by smearing the entrails of a caterpillar on the arrow point, "the action of which has a generic resemblance to snake poison." Other tribes used the amaryllis disticha, various species of Euphorbium and Acokanthera, alone or mixed with snake, spider, and beetle poisons.

The Choco Indians, in Colombia, South America, also use a peculiar poison, derived from a tree-frog, the *Phyllobates chocoensis*, which they hold on a stick near a fire, when the heat causes the glands of the skin to secrete the poisonous fluid. The Choco Indian poison is innocuous when given by the mouth; a few experiments have been made with it in France, but its exact action remains rather doubtful. It is capable of killing large carnivora.

The most deadly are the arrow poisons derived from the root wood of different species of *Acokanthera*, trees about fifteen feet high—*A. schimperi*, *A. deflersii*, and *A. ouabai*. From these is prepared the deadly arrow poison of the Somalis known now for a long time and very fully described by Burton ("First Footsteps in East Africa," 1856). The poison is known as Waba, Wabayo, or Ouabaio. It is a thick tar-like, watery extract, the active constituent in which is a glucoside known as ouabain (Arnaud), and it is made by splitting up the root into small pieces, boiling these with water, inspissating the juice, and then adding usually snake-venom or other poisonous vegetable extracts. Burton says that cattle eat the leaves of the tree only if very hungry, and that the berries are edible.

Besides the Somalis, the Wa Nyika Wakamba, the Massai Wa Nyamwesi, and many tribes of Eastern and Central Equatorial Africa use practically this same poison, although there are many minor differences in its composition among these different peoples. Many of the prepared poisons contain very irritating substances, which are productive of severe local symptoms in the wounded. Boehm states that the poisonous dose for a dog per kilo. of its weight is about $\frac{1}{10}$th grain echujin, $\frac{1}{250}$th grain strophanthin, and $\frac{1}{150}$th grain ouabain, which gives some idea of their extreme toxicity.

In South-West Africa the Ovambas also use a heart poison derived from a species of *Adenium*, while the *Strophanthus* is widely used on the Congo, on Lake Nyassa, the Zambesi, Gaboon, Guinea, Cameroons, and Senegambia. Various *Euphorbias* and other imperfectly known plants are also largely employed.

Mr. Crawford Angus gives a graphic account of the poisons used in Azimba and Chapitaland in Central Africa. The natives use an arrow, the slightest scratch of which causes death, the poison being known only to certain chief men who collect it and serve it out to the others.

The arrow poison of the Pigmies is a mixture of a cardiac poison and strychnine poison, with some others. It is very deadly, and one arrow will kill an elephant, but Stublman states that in man, if the head be at once extracted and the wound scraped and washed, fatal consequences are frequently averted.

[1] *Pharm. Jour.*, 1898, pp. 550 and 585.

Another set of arrow poisons which have a similar action on the heart are those made from the juice of the famous upas tree, the *Upas antiar*, growing in Borneo, Java, and adjacent parts. It is a very large forest tree, and the poisonous sap is obtained from incisions made into the bark. The active principle—a crystalline glucoside called antiarin—is extremely poisonous, and experiments which I made with it showed that ·000015 gramme was sufficient to kill an ordinary-sized frog in comparison with ·00022 gramme strophanthin and ·00037 gramme urechitin. The sap is known as Ipoh Kayu (tree poison) among the natives. It kills guinea-pigs and other small animals in a few minutes from stoppage of the heart, and has been used in Cochin China against the French soldiers, who died in from half an hour to several days after receiving their wounds. It is in use throughout the Eastern Archipelago by nearly all the native peoples, pure, or mixed with snake-poison, scorpions, centipedes, other plants, and occasionally with arsenic. The different prepared poisons vary greatly in strength, and one old specimen which I examined was quite innocuous.

Aconite root (*A. ferox*), under the name of Bis, Bish, Bikh, and sometimes called tiger poison, is used as an arrow poison in Nepaul and along the eastern frontiers of our Indian Empire, and on the French and Chinese frontiers also most probably. It is very active, but the effects of aconite are so well known that I need not linger over them here.

We come lastly to the different species of *Strychnos*, which are so largely used in South America, in the East Indian Archipelago, and to a much more limited extent in Africa for preparing these poisons. The most famous of them is *Curare*, first brought to Europe in 1595 by Sir Walter Raleigh. Under various names it is used over the immense tract of country comprised in the basins of the Amazon and Orinoco and their tributaries. A very minute and interesting account of its manufacture has been given by Humboldt, from which it appears that it is a concentrated extract made with cold water from the bark of several species of *Strychnos*, and that this is mixed with other poisonous and non-poisonous ingredients to increase its efficacy and consistence. It is not poisonous when swallowed, owing, it is said, to the slow rate at which it is absorbed, and Humboldt says that the Indians lick it off their fingers and use it as a stomachic tonic. Its harmlessness when given by the mouth has been frequently confirmed by exact experiment. But when injected subcutaneously it proves rapidly fatal by paralysing the ends of the motor nerves in muscle, so that movement becomes impossible and death takes place from the respiratory muscles ceasing to act on the chest wall. A large dose kills in a few minutes, and there is no antidote known. Besides this action on the nerves, which is due to curarin, it has a paralysing effect on the heart, due to a second active principle, discovered by Boehm and named by him curin.

In the Malayan Archipelago the *Strychnos* or *Upas tieute* furnishes a sap largely used for poisoning arrows, and, the active principles being strychnine and brucine, we get the well-known convulsant effect of these substances in animals or men struck by the arrows.

I have, however, examined the root bark of two species of *Strychnos* used as an arrow poison by the natives of Perak in the Straits Settlements, and found that both had a marked digitalis-like action on the heart, as well as a curare-like action on the motor nerves.[1] These are mixed with a third substance called "prual," which paralyses the muscles. When these different ingredients are mixed they form a most efficient means of dealing death, seeing that they paralyse simultaneously the heart, the motor nerves, and the voluntary muscles.

In conclusion, I may just mention two other poisons, neither of which is perhaps thoroughly authenticated. The Ainos in Japan are said to use a preparation made from aconite and tobacco, while the natives of the New Hebrides are stated to smear their arrows and spears with damp earth containing the tetanus bacillus, so that a cut infects their victim with this disease. It is more probable, perhaps, that the wound inflicted by these weapons sometimes becomes infected with the bacilli through the ordinary channels. The North American Indians do not use arrow poisons, nor do the aborigines of Australia, so far as is known.

In spite of the large number of arrow poisons which are known to us, the toxic actions are not very numerous, and can be roughly classed under five headings (although this does not include all, especially locally irritating substances): (1) Those which act on the heart and muscles, like digitalis; (2) those which act

[1] *Lab. Rep. Roy. Coll. Phys. Ed.*, vol. 6.

on the nerve-endings, like curare ; (3) those which act on the nervous system and heart, like aconite ; (4) those which act on the spinal cord, like strychnine ; and (5) those which have an action something like snake-poison.

The use of arrow poisons in Tanganyika is discussed by Raymond[1] The most common arrow poison there is Ouabain, the lethal dose of which is stated to be 2 mgr. for a ten-stone man.

On an antidote to these poisons, the following paragraph appears in the *Pharm. Jour.* for 1897, p. 458 :—" The attention of the Government has recently been directed to the subject of arrow poison by the fatal results following the wounds caused by poisoned arrows in Uganda, and specimens of the arrows with the poison on them have, it appears, been sent by the Marquis of Salisbury to the Royal College of Physicians for investigation, in the hope that some antidote might be devised. The chief poison used in this part of Central Africa is evidently an *Acokanthera*,[2] and an antidote to its action would in all probability prove an antidote to the poison of the arrows. Thanks to the researches of Professor T. R. Fraser, of Edinburgh, we are now in possession of a knowledge of the physiological action of the plant, and it should not be difficult to find a physiological antidote. Meanwhile, however, news has arrived from Uganda that Dr. Macpherson, who was with the Grant Column on the way to the Man Mountains, west of the Uganda Road, has discovered that the injection of a solution of strychnine answers the purpose of an antidote. He was able to bring round to life men wounded with the poisoned arrows, where previously the wounds had generally proved fatal. Should the investigations made by the College of Physicians prove that Dr. Macpherson's remedy is entirely successful, the fact will probably prove useful in two ways, for another antidote to strychnine poisoning will have been added to those already known, if, as may be presumed, the drugs should be mutually antidotal. Other African arrow poisons contain *Strophanthus*, and as strophanthin belongs also the class of cardiac poisons and to the same natural order, it would be important to determine whether strychnine is also antidotal to *Strophanthus*."

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Poisoning by Artemisia (Sp. Var.). Oil of Wormwood. Absinthe

Source and Method of Occurrence. The chief constituent of the oil is a camphor-like body known as Thujol, which acts in a manner similar to camphor, namely excitation of the central nervous system followed by unconsciousness and general convulsions first chronic then tonic in nature.[3] Absinthe was formerly prepared from a species of artemisia and other oils and was much used in France where it caused, according to Legrand, derangement of the digestive organs, intense thirst, restlessness, giddiness, tingling in the ears, and illusions of sight and hearing

[1] *East African Med. Jour.*, 15 : 419 (1939).
[2] See *Pharm. Jour.* [3], vol. 24, p. 41.
[3] Sampson, W. L., *Jour. Pharm. and Exp. Therap.*, 65 : 275 (1939).

These symptoms were followed by trembling in the arms, hands and legs, numbness in the limbs, loss of muscular power, delirium, loss of intellect, general paralysis, and death.

Magnan, who had under his observation 250 patients more or less injured in health by the abuse of this intoxicating liquid, and who has besides performed numerous experiments on animals, states that epileptic convulsions are generally observed in these cases. *Delirium tremens* is the ordinary result of the abuse of alcohol, but the epileptic attacks are specially referable to the absinthe. Magnan describes it as "absinthe epilepsy."[1]

At the present time, absinthe from reputable makers is free from thuyon.[2]

The following is the report of a case of poisoning by oil of wormwood :

A druggist's shopman was found early one morning by his master, lying on the floor of the shop, perfectly insensible, convulsed, and foaming at the mouth. He was in a short time no longer violently convulsed, but was still insensible ; the jaws were clenched, and the pupils dilated. The pulse was weak, compressible, and slow. From time to time he uttered incoherent expressions, and attempted to vomit. Repeated doses of stimulants, sal volatile and water, lime water, and an emetic of mustard and sulphate of zinc were administered. Free vomiting ensued, and consciousness partially returned. Artificial warmth was applied to the limbs, and brandy given at intervals, with draughts of milk and lime water. He gradually recovered. The matters vomited smelt strongly of oil of wormwood, and the nature of the poison was placed beyond doubt by the discovery of the bottle, with marks on its mouth of the oil having been recently poured out. The druggist stated that at least half an ounce had been taken. From the persistent smell of the oil in the ejected matters, after repeated vomiting, it is probable that this was even less than the real quantity. The man, on recovering, had totally forgotten all the circumstances connected with the case, and persisted in stating that he knew no reason why he should have taken it.

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Poisoning by Artemisia maritima

Source and Method of Occurrence. The dried unexpanded heads of the plant, which is not uncommon on the coasts of the British Islands, form santonica, or wormseed, from which a naphthalene derivative known as santonin is obtained ; it is much used as a vermifuge, and cases of poisoning from idiosyncrasy and overdoses are known. Santonica contains from 2 to 2½ per cent. of santonin.

Santonin has been occasionally mixed with and mistaken for strychnine or *vice versa*.

Toxicity and Fatal Dose. The toxicity of santonin must be reckoned as rather high, for about two grains have proved fatal to a child, aged five and a half years, in twelve hours. Recovery in a child has, however, followed after taking ten grains. Recovery has taken place, in a man, after taking an ounce of santonin in mistake for Epsom salts.

[1] Husemann's *Jahresbericht*, 1872, p. 499, and Bouchardat's "Ann. de Thérap.," 1872, p. 66.
[2] *J. A.M. A.*, 113 : 1822 (1939).

The official dose is one to three grains, and for a child a year old about one-third to one-half of a grain should suffice.

Symptoms. (i.) The special symptom is a peculiar disturbance of vision, objects appearing at first blue, then yellow; finally colour-blindness results for some time. This curious fact is explained by Hufner and Helmholtz as being due to the action of the santonin on the visual purple of the retina. (ii.) Headache, giddiness and a species of intoxication frequently occur. (iii.) Gastric pain, vomiting, and laboured respiration may be produced. (iv.) In large doses convulsions, stupor, loss of consciousness, and death from collapse occur. (v.) The urinary secretion is increased, and the urine is coloured saffron-yellow, which turns pink if the urine is made alkaline; in severe poisoning there may be hæmaturia.

Treatment. The stomach should be emptied and washed out by means of the stomach tube, or an emetic of mustard and water should be given, and a suitable cathartic should be given. Stimulants should be administered to counteract the depressing action of the poison, and if convulsions are present, they should be treated with potassium bromide and chloral, or by short-acting barbiturates given intravenously.

Post-mortem Appearances. Nothing characteristic.

Analysis. Santonin may be extracted from *acid* aqueous solutions by shaking out with chloroform; it will not come away from alkaline solutions, as it plays the part of a weak acid, forming combinations with alkalies which are soluble in water.

A solution of sodium hydroxide produces a violet-red colour with santonin. Dragendorff has devised a modification of a former test which is thus performed: A little sulphuric acid diluted with half its volume of water is added to some santonin and gently heated until a yellow colour is produced; when cold a few drops of a very dilute solution of ferric chloride are added, and on again warming a blue or reddish violet colour is produced (Mann).

Luff thus describes the sulphuric acid test:—

Strong sulphuric acid is diluted with half its bulk of water, a little of the diluted acid is added to some santonin in a porcelain dish, and the mixture warmed on a water-bath until a yellow colour is developed: while warm a few drops of dilute solution of ferric chloride are added drop by drop: as each drop falls in, a ring of a beautiful red or reddish-violet colour is produced around each drop, changing to purple, and afterwards to brown. The colour is soluble in amyl alcohol.

The presence of santonin in the urine may usually be ascertained by the addition of a little sodium hydroxide: if present a red colour is produced. Rhubarb present in the urine yields the same reaction: but if, after the addition of sodium hydroxide, excess of milk of lime is added, and the urine afterwards filtered, the filtrate is colourless if the reddening is due to rhubarb, but retains its colour if it is due to santonin.

Santonin crystallises in four-sided tables, which have the remarkable property of acquiring a brilliant yellow colour by exposure to light (*photosantonin*). It is only very slightly soluble in cold water, but more

so in hot water, when it gives an almost tasteless solution; but it is soluble in alcohol, and the solution has a bitter taste. Its best solvent is chloroform, four and a half parts of which dissolve one part. It melts at 169° C. to 171° C., and sublimes in white crystals a few degrees above its melting point. Nitric, iodic, and sulphuric acids have no action on it in the cold. Sulphomolybdic acid produces a pale reddish-brown colour. Bichromate of potash added to the mixture with sulphuric acid produces no colours like strychnine, but only green sulphate of chromium.

Cases. A man gave to his daughter, *æt.* 10, about 155 grains of wormseed for the cure of worms. Within two or three hours she was seized with violent vomiting, followed by convulsions, with coldness of skin. The following day worms were discharged. She was seen by a doctor on the third day. There was severe vomiting, with convulsions, the pupils were dilated and insensible to light. The girl was drowsy, and suffered from some pain in the stomach. She died before any medicines could be employed. The body was not inspected.

Mann records the following curious case of chronic poisoning by santonin :—

A boy aged eleven years, who, on account of pain in the abdomen, which was supposed by his mother to be due to worms, had santonin given to him for months. Clonic spasms then developed, to combat which the doses of santonin were increased. Paralysis, twitchings, dizziness, pain in the head, vomiting, yellow and violent vision, sparks before the eyes, and finally loss of speech occurred and necessitated medical advice. Under treatment the patient was able to walk in six weeks, but it was nine weeks before he regained the power of speech.

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Poisoning by Arum maculatum (Araceæ)

Source and Method of Occurrence. This plant is a common British plant, putting up its leaves and spathe in early spring. The leaves are glossy and spotted, and about August clusters of scarlet berries appear. It is popularly known as " lords and ladies," " cuckoo-pint," " water robin," etc., and children occasionally suffer from chewing parts of the plant. During the potato famine in Ireland large quantities of the big bulbous root of the plant were dug up and eaten. When eaten raw they produced toxic symptoms, but after boiling they appeared to be innocuous. The plant contains an acrid juice of unknown chemical nature which acts as a powerful irritant.

A girl, aged three years, died suddenly after having eaten some plants in the garden. A *post-mortem* examination showed that death was due to poisoning with the leaves of the Arum maculatum.[1]

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[1] *Lancet*, 1900, 1, p. 1176.

Poisoning by Aspidium filix-mas, or Oil of Male Fern

Source and Method of Occurrence. The official preparation, Extr. filicis liquidum, dose 45 to 90 minims, is obtained from the rhizome of the ordinary male fern *aspidum filix-mas*. The fern is common in England. Cases of poisoning by it are rare, but fatal cases have been recorded.

Toxicity and Fatal Dose. The medicament depends for its active properties chiefly on the presence of filicic acid, a derivative of phloroglucinol, which should be present to the extent of 24 to 26 per cent. by weight.

The toxicity is very variable, probably as much because of variation in the rate of absorption as of varied individual susceptibility. It has been said, though without much supporting evidence, that the toxicity of male fern is increased by the simultaneous administration of oily cathartics such as castor oil. Many deaths have occurred when the dose has exceeded 2 drachms of the oleoresin.

Symptoms. With toxic doses there may be delirium, cramp, convulsions and coma, death occurring from respiratory failure. Blindness, temporary or permanent, has been reported.

Treatment. If spontaneous vomiting does not occur, the stomach should be emptied either by the tube or an emetic, after which general treatment will be required, and probably the administration of stimulants.

Case. Dr. C. F. Wakefield reported the following case which occured in 1898 :—

A man, *æt.* 25, at 10.30 a.m., took eight ten-minim capsules of oil of male fern ; at 11.30, Epsom salts, one ounce, at once ; 3.5 p.m., attended his cows ; 8.10 p.m., at the inn, where he drank two pints of beer only. He had no food all day ; he walked home, half a mile, shut up his chickens and poultry for the night and talked to his wife quite rationally. He then, about 10.30, stooped down to take off his boots, and was sick two or three times, vomiting " half a pailful." Bowels had been open six times during day. 10.30–12.30, he was quite unconscious ; before becoming so five men held him down to prevent him biting. I washed him out, but only got away a lot of glairy mucus. He came round about 1 a.m. Pulse 140 ; feet cold. He had previously taken eighteen five-minim capsules followed by castor-oil immediately. " It all went through in half an hour." The chemist sold the box of three dozen capsules without any directions or advice.

The following cases are related by Mann [1] :—

A man, thirty years old, was given a draught containing one ounce and a half instead of one drachm and a half of the extract of male fern, which he took in two doses. Soon after the first dose he felt unwell, and after the second, which was given some hours subsequently, he began to vomit, and was purged ; then followed cramps, profuse sweating, delirium and coma, which ended in death about twenty hours after the draught was taken. At the necropsy the omentum and the peritoneal covering of the small intestines were bright red, and in the sub-mucous tissue of the stomach were ecchymoses with linear extravasations on the surface of the mucous membrane. An instructive case is related by Freyer in which a child, aged two and three-quarters, took eight capsules—each containing about fifteen grains of extract of male fern, along with the same quantity of castor-oil—in five hours ; she became somnolent and as though paralysed, and died after the occurrence of some spasms. Section showed petechial ecchymoses in the mucous membrane of the stomach, pronounced injection of the mucous membrane of the intestines and venous filling of the various organs. The interesting point to note

[1] " For. Med."

is that three weeks previously the child took double the quantity of the extract, *but without the castor-oil*. A case is recorded by Hoffmann, in which a child, five and a half years old, had very nearly two drachms of the extract given to her in three draughts; death took place in six hours, with symptoms of trismus and general spasms. The same appearances were found as in the other cases.

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Poisoning by Atractylis gummifera

Source and Method of Occurrence. This plant seems to be a native of the Mediterranean district and grows freely in Malta. The following case is reported by Dr. Zammit, from the Public Health Laboratory, Malta, in the *B.M.J.*, 1898, 1, p. 211.

The carline thistle (local name, Xeckit-il-Miskta) is the *Carlina gummifera* (Dec.), *Atractylis gummifera* (Lin.), a plant common in Malta and in other countries on the shores of the Mediterranean. It is notable for its root, which can grow to enormous proportions, and for its purple composite flower surrounded by spinous leaves.

Three children ate the root of one of these plants, which they cut with a knife; two of them, a girl aged eight years and a boy aged nine, eating it rather freely. They ate it on a Wednesday afternoon, and did not complain at all before the catastrophe came on. On Thursday evening the girl was taken ill, and the parents were soon alarmed at the stertorous breathing and at the drowsiness of the child. There was some retching, but no actual vomiting. Early in the morning the child became collapsed and died before any medical aid could be got. In the evening of Friday the boy returned home and refused to eat. He was told that the girl had died, and he confessed that they had been eating some roots the day before. He went to bed, and soon after the family were startled at his difficult breathing. The father of the girl (the children were cousins) was sent for, and he declared that his daughter had suffered in the same way. The district medical officer was hastily called, but it was too late, as the boy died a few hours after, comatose and with marked signs of asphyxia.

At the *post-mortem* examination the two bodies had the same appearance. The tongue protruded slightly between the lips; the pupils were greatly dilated. The appearance consisted in a general congestion of the venous system. In the girl all the organs were highly congested, the lungs and the brain especially so. The stomach showed capillary injection in limited areas. It contained a brown liquid in which fibres of the ingested root were identified. The intestines were congested and contained abundant solid fæces. The heart in both cases was flaccid, and contained a little dark fluid blood.

In the boy the congestion was not so well marked in the organs, with the except of the brain, of which the veins were injected to the extreme. No trace of the root could be found in the stomach, but the fibres were found abundantly in the intestines, which contained a very great quantity of solid fæces. No attempt was made to examine chemically the organs, as the poisonous principle of the plant is not known to me. I have searched all the books of botany and medical jurisprudence which I could find here, but no cases of poisoning by this plant are fully described, nor is the active principle of the plant mentioned anywhere.

According to Rho, *A. gummifera* is a common abortifacient among the Arabs. Its action is somewhat slow, but is often fatal with vomiting, colic, anuria, dyspnœa, convulsions and coma.

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Poisoning by Atropa belladonna (Solanaceæ)

Source and Method of Occurrence. All parts of this plant, *Atropa belladonna* (also known as deadly nightshade), are poisonous. The fruit closely resembles black cherries, and accidents from children eating the fruit are known. The root, administered in the form of decoction as an enema, has destroyed life. Eighty grains of the root were employed, and the liquid, strained and reduced by evaporation to four ounces, was injected. After a slight stage of excitement, death took place in five hours.

The preparations of belladonna are: the leaves, containing 0·3 per cent. of alkaloids, from which the tincture and dry extract are prepared, and the root, containing 0·4 per cent. alkaloid, from which the plaster, liquid extract and liniment are prepared. These preparations contain the following percentages of alkaloids, calculated as hyoscyamine, Tinct. 0·03 per cent. (dose 5–30 minims), Ext. Sicc. 1·00 per cent. (dose $\frac{1}{4}$–1 grain), Ext. Liq. 0·75 per cent. (dose $\frac{1}{4}$–1 minim), Liniment 0·375 per cent.

There is also a plaster containing 0·25 per cent. of alkaloids and a suppository containing in each $2\frac{1}{2}$ minims of liquid extract; an ointment and lamellæ, each containing $\frac{1}{5000}$ grain atropine, for eye work.

All parts of the plant contain the alkaloids atropine, hyoscyamine and hyoscine in varying proportions, and the toxic effects are due to the action of these alkaloids. In addition to Atropa belladonna, these alkaloids are found in various species of datura, hyoscyamus, scopolia, the mandrakes (mandragora) and many other members of the solanaceæ. The crude drugs from different members of this family contain different relative amounts of atropine hyoscyamine and hyoscine, and therefore the effects may vary in degree though not in kind. Atropine and hyoscyamine are isomeric, their action is identical, but hyoscyamine has a more intense peripheral action. Hyoscine or scopolamine, however, has rather different action, especially on the central nervous system.

Toxicity and Fatal Dose. The active principle (atropine) to which all the preparations owe their toxic effects is extremely poisonous, the official dose being from $\frac{1}{240}$ to $\frac{1}{60}$ of a grain. Severe symptoms may occur after as little as $\frac{1}{8}$ grain but the usual toxic dose may be put at about $1\frac{1}{2}$ grains.

A woman, *æt.* 66, swallowed a teaspoonful of belladonna liniment, and, after exhibiting the usual symptoms, died within sixteen hours. This is the smallest recorded fatal dose of the liniment. Recovery is recorded[1] from half an ounce of the extract. **Half a grain** of atropine has proved fatal. A case is recorded of recovery from a grain of atropine after treatment with morphia. We have seen a case in which recovery took place after fifteen grains of atropine taken by the mouth, but this must be extremely rare.

[1] *Lancet*, 1891.

Susceptibility to the action of the drug varies enormously in different people. It varies also in animals, rabbits being remarkably immune to its effects.

Duration. The symptoms come on within a few minutes to an hour or so, depending on the method of administration, but usually run a long course, and unless large doses are taken recovery is the rule.

Symptoms. Shortly after taking the drug there is a feeling of dryness in the mouth and throat, and talking and swallowing become difficult, due to the inhibition of the secretions. The face is flushed scarlet, and, especially in children, a rash may appear over the face, neck and upper parts of the trunk with, later, desquamation. The pupils are dilated, so that vision becomes indistinct, and they do not contract with light. Accommodation is paralysed. The temperature is often raised—sometimes alarmingly so in young children. The pulse is accelerated by the paralysis of the vagus nerve, and in many cases is uncountable. The respirations are increased. The patient may pass through a stage of excitement due to the stimulation of the cortex. He becomes restless, talkative, and may be delirious. Sometimes he is wildly excitable and destructive. In his delirium he is liable to make co-ordinated purposive movements, such as attempting to thread an imaginary needle. Giddiness, with staggering gait and numbness occur. He passes from this stage to one of depression of the cortex, and he becomes sleepy. His sleep becomes deeper and passes into coma. The respirations may become slow and shallow owing to the depression of the medulla, and death may take place from respiratory failure. If recovery takes place it is comparatively slow, and the patient remains in a bemused state for twenty-four to thirty-six hours. The dilation of the pupils may be observed for several days, and sometimes continued delerious symptoms as well. Quite often the patient shows signs of irritability and intolerance.

Hyoscine or scopolamine has peripheral effects similar to those of atropine, but the action on the secretions and on the oculomotor are stronger and the action on the vagus weaker. The central action differs inasmuch as hyoscine action is mainly sedative. Doses from $\frac{1}{60}$–$\frac{1}{20}$ grains cause drowsiness leading to a deep sleep in a matter of moments. The preliminary stage of excitement is very short and seldom observed. With toxic doses there may be a period of excitement and delirium, but the patient soon passes into stupor and coma[1] and dies from asphyxia.

The following cases are typical of the symptoms :—

A boy, *æt.* 14, ate, soon after breakfast, about thirty belladonna berries, which he had bought as fruit in the streets. Within about three hours he had the sensation of his face being swollen ; his throat became hot and dry, his vision was impaired, objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and the eyelids swollen ; there were occasional flashes of light before his eyes. He tried to eat, but could not swallow on account of the state of his throat. In endeavouring to walk home he stumbled and staggered ; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated ; he was incoherent—frequently counted his money, and did not know the silver from the copper coins. His eyes had a fixed, brilliant, and dazzling gaze ; he could neither hear

[1] McCowan, P. K., *et alia*, *B.M.J.*, 1 : 779 (1926).

nor speak plainly and there was great thirst ; he caught at imaginary objects in the air and seemed to have lost all knowledge of distance. His fingers were in constant motion : there was headache, but neither vomiting nor purging. He did not reach the hospital until nine hours had elapsed, and the symptoms were then much the same as those above described. He attempted to get out of bed with a reeling, drunken motion : his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris to be seen, and the eyes were quite insensible to light. The eyelids did not close when the hand was passed suddenly before them. He had apparently lost the power of vision, although he stared fixedly at objects as if he saw them. The nerves of common sensation were unaffected. When placed on his legs he could not stand. His pulse was ninety, feeble and compressible ; his mouth was in constant motion as if eating. On admission his bladder was full of urine. He continued in this state for two days, being occasionally conscious, when by a free evacuation of the bowels some small seeds were passed : these were examined and identified as the seeds of belladonna. The boy gradually recovered and left the hospital on the sixth day after his admission : the progress of recovery was indicated by the state of the pupils, which had only then acquired their natural size and power of contraction. In three other cases which occurred at the same time, the fruits having been baked in a pie, pains in the limbs, drowsiness, insensibility, and convulsions were among the symptoms. In two instances of poisoning by the fruits, the symptoms bore some resemblance to those of delirium tremens. The symptoms are sometimes delayed.

In September 1892, a boy, aged five years, died at Mortlake from eating belladonna fruits. In this case the symptoms did not appear till the morning after the fruits were supposed to have been eaten, and he lived sixteen hours after the appearance of symptoms of atropine poisoning. There was no delirium ; there was thirst, flushing of the face, and the heart's beats were audible at a distance of several feet. Unconsciousness quickly supervened. The seeds of the plant were found in the intestines after death, and atropine both in the stomach contents and in the urine was detected.

Two persons swallowed a small spoonful of the *extract* of belladonna by mistake for that of juniper. There were speedily indistinctness of vision, tottering gait, delirium, incoherency hallucinations, and dilatation of the pupils. In one there was great cerebral excitement. Under treatment, the symptoms of poisoning disappeared in two days : but one of the patients died on the seventh day from disease. In a case of compound poisoning by extract of belladonna and tincture of opium there was the violent excitement produced by belladonna, but the pupils were strongly contracted as in poisoning by opium.

Treatment. The stomach should be emptied and thoroughly washed out by means of the stomach tube, using an alkaloidal antidote, but not permanganate of potash, which is ineffective against atropine. The tissues are extremely dry in atropine poisoning, hence it is essential to lubricate the tube before trying to pass it. An emetic of mustard and water should be given if no tube is at hand. If the plant or seed has been ingested, the bowel must be washed out frequently to get rid of all vegetable matter. In the excitable stage it may be necessary to use sedatives, but very great care must be exercised owing to the danger

of paralysing the medullary centres. Luminal may be used or, preferably, chloroform or ether, as they are more under control. Pilocarpine has been used as an antidote, being said to increase the patient's comfort and to help in relieving the usual symptoms, though it does not antagonise the action of atropine on the brain. In the collapse stage, stimulants and hot coffee should be administered, and artificial respiration or inhalation of carbon dioxide and oxygen may be found necessary. Caffeine is of value as a stimulant. The administration of morphine hypodermically has been frequently recommended, and certainly the tolerance of morphine exhibited in atropine poisoning is striking, and is in favour of the antagonism of these drugs ; but although atropine may be of use in cases of opium or morphine poisoning, the converse is not by any means so certain. Morphine by its depressant action on the respiratory centre may be actively dangerous, and there is some reason to believe that hyoscine and morphine produce additional narcosis. An interesting example of the effects of pilocarpine is related in which a male, aged sixty, had administered to him by mistake a tablespoonful of a liniment consisting of equal parts of belladonna liniment and tincture of opium. Although the so-called physiological antidote morphine, was present in this liniment, the typical symptoms of belladonna poisoning supervened. Recovery took place after hypodermic injections of pilocarpine, which were repeated until one grain of pilocarpine in all was injected ; the patient lapsed into a comatose condition during an interval of about six hours, whilst a fresh supply of pilocarpine was being obtained.

The Edinburgh Committee, presided over by Dr. Hughes Bennett, concluded : (1) that sulphate of atropine is, within a limited range, physiologically antagonistic to meconate of morphine : (2) that meconate of morphine does not act antidotally after a large dose of atropine, and thus, while atropine is an antidote to morphine, morphine is not an antidote to atropine. Physostigmine, in relation to atropine, is considered to have a wider range of antidotal action than morphine. Although it is experimentally proved that atropine can avert death from physostigmine poisoning, the question yet remains unsolved whether physostigmine can prevent death from atropine.

Post-mortem Appearances. Unless portions of the plant such as the seeds or leaves are found in the stomach, there is little else to be observed. The congestion is simply due to cardio-respiratory failure, common to many deaths other than poisoning. The pupils have been found dilated many hours after death.

In some instances the mucous membrane has been dyed by the juice of the fruit.

Analysis. The greater part of the atropine rapidly disappears from the blood, and is mostly excreted in the urine. The indigestible nature of the leaves, fruit, and seeds will commonly lead to their detection in the matters vomited or passed by the bowels, or in the contents of the viscera after death. The *seeds* of belladonna are small but are readily identified. They are of a somewhat oval shape, and of a dark colour. Under a low magnifying power they have a honeycombed surface. In henbane the surface of the seeds presents more irregular depressions.

[1] Cushny, "Pharmacology," 1925.

resembling those seen on certain corals or madrepores. The colouring matter of the belladonna fruit is of a deep purple hue. The leaves are known by their botanical characters, and a decoction or infusion of them by the liquid causing dilation of the pupil.

The urine, blood, or other organic liquid containing this poison, applied to the eye of an animal, causes dilation of the pupil, though the delicacy of the test is decreased by the presence of inorganic matter in considerable amounts. This may be employed as a physiological test for detecting the presence of atropine in an extract made from the contents of the stomach, or of any organic liquid. It is perhaps the most delicate test for the substance, 0·00001 milligram producing definite dilation.

In extracting the atropine alkaloids from tissues, etc., by the Stas-Otto process, great care must be taken to avoid hydrolysis. If it is important to know which particular alkaloid is present, the use of alkalies (which cause racemisation) must be reduced to a minimum. For the final extraction, chloroform is the best solvent.

Atropine is a white crystalline substance, not very soluble in water, but easily dissolved by alcohol, ether, and dilute acids. It does not readily crystallise, but it forms crystallisable salts. Ammonia added to the solution of sulphate of atropine does not separate the alkaloid in distinct crystals. In this respect it differs from morphine and strychnine. When atropine is heated on platinum it melts, darkens in colour, and burns with a yellowish smoky flame. Sulphuric, hydrochloric, and nitric acids dissolve it without any change of colour. Water added to the mixture of sulphuric acid produces no change ; but a crystal of bichromate of potassium produces a green colour. Tannic acid precipitates the alkaloid from its solutions ; but the most effective precipitant is potassio-mercuric iodide, which throws down a dense white precipitate even in very dilute solutions. Atropine is also precipitated by auric chloride, but, unlike strychnine, it is not precipitated by sulphocyanide of potassium nor by chromate of potassium.

The three important alkaloids of belladonna are closely related. Both atropine and hyoscyamine are esters of tropic acid, $C_9H_{10}ON_3$ and tropine, $C_8H_{15}ON$. Hyoscyamine is lævo-rotatory, and is readily converted (*e.g.*, at a temperature slightly above its melting point, or when its alcohol solution is made slightly alkaline and allowed to stand) to a racemic mixture which is atropine. Hyoscine (*l*-scopolamine) is a lævo-rotatory ester of tropic acid and scopine, $C_8H_{13}O_2N$; it bears the same relationship to atroscine that hyoscyamine does to atropine.

(1) *Appearance.* Atropine is deposited, on evaporation of its chloroform solution, in lustrous needles, melting at 117° to 118° C. ; hyoscyamine in rosettes of slender needles or as a crystalline powder of indeterminate form, melting at 107° C. ; hyoscine as a syrup.

(2) *Aurichloride.* Atropine aurichloride is deposited, on addition of gold chloride to a dilute solution of the alkaloid in water acidified with HCl, as an amorphous or oily precipitate which gradually becomes crystalline, and melts under boiling water (the dry salt is lustreless and melts at 137° to 139° C). Hyoscyamine aurichloride is deposited in golden yellow scales which do not melt under water, retain their lustre when dry, and melt at 165° C. Hyoscine aurichloride forms crystalline

needles, which do not melt under water, and, when dry, melt at 204° to 205° C.

(3) *Picrates.* Atropine picrate crystallises in rectangular plates, of melting point 175° to 176° C.; hyoscyamine picrate in quadrangular plates or crusts of needles, melting at 165° C.; and hyoscine in fine needles, melting at 187° to 188° C. Hyoscine picrate crystallises only very slowly.

(4) *Vitali's Test.* A trace of atropine, hyoscyamine, or hyoscine (0·0001 mgrm. is said to be enough) is dissolved in a drop of fuming nitric acid, and the solution is evaporated on a boiling water-bath. The residue is touched with a drop of freshly prepared alcoholic potash, when a deep violet colour appears, changes slowly to dark red and ultimately disappears (but can be restored by addition of more alcoholic potash). Alkaloids, such as morphine, which give a strong coloration with nitric acid alone, interfere with the test, but of sixty alkaloids examined, only the three belladonna alkaloids were found to give a violet colour under the conditions; strychnine gives a yellow which deepens to reddish violet, brucine a greenish colour, and homatropine [1] a yellow.

(5) *Phenolphthalein.* A trace of the alkaloid is placed on a piece of filter paper previously soaked in an alcoholic solution of phenolphthalein and dried. The paper is then wetted with alcohol. In presence of *free* atropine, hyoscyamine, or hyoscine, no colour is produced, but if the alcohol is allowed to evaporate and the paper is then touched with a drop of water, a red coloration appears. This colour is destroyed by alcohol, but reappears when the alcohol is evaporated and replaced by water. The reaction is given by homatropine, by coniine and nicotine (according to Plugge) but not by any other common alkaloid.

(6) *Gerrard's Test.* A 2 per cent. solution of mercuric chloride in 50 per cent. alcohol is gradually added to a little of the free alkaloid in a watch glass (Gerrard suggests $\frac{1}{10}$ grain of the alkaloid and 20 minims of the $HgCl_2$ solution—excess of the reagent must be avoided). Atropine yields a red coloration at once; hyoscyamine gives a yellow colour which darkens a little, and turns red on heating; hyoscine gives neither red nor yellow. If the amount of alkaloid is large, precipitates of the appropriate colour are formed. Many other alkaloids give white precipitates, which in some cases (morphine, codeine) become pale yellow on heating, or red on standing (cocaine).

(7) *Wormley's Test.* Solutions of atropine or hyoscyamine or their salts (1 : 10,000) give, on addition of a saturated solution of bromine in hydrobromic acid, yellow amorphous precipitates which soon become crystalline. Though many other alkaloids give amorphous precipitates, Wormley considered the crystalline precipitate characteristic of these two. A syrupy solution of hyoscine, he states, gives reddish-yellow globules which later crystallise, but not crystals from solutions weaker than 1 per cent. Carr, however, has obtained crystalline precipitates from 0·05 per cent. solutions of hyoscine and also from solutions of homatropine, scopolamine, and caffeine.

[1] A synthetic substance, the mandelic ester of tropine, mydriatic, much less toxic than atropine, used in ophthalmic surgery.

(8) *Mydriatic Test.* A drop of a neutral solution is instilled into one eye of a cat which is kept in the dark for half an hour and then examined in bright sunlight. The atropine group of alkaloids give a marked dilation of the pupils. Other substances with (less) mydriatic activity do not respond to Vitali's test.

Cases. The following case [1] illustrates the symptoms of poisoning by atropine.

At 2.30 on the morning of March 7th, 1901, I was called to attend a boy, aged five years, alleged to have been accidentally poisoned by his mother. At 9.30 p.m. the mother gave the child a large tablespoonful of what she took to be syrup of senna. She then left home to nurse a sick relative, and did not return till shortly before 2.30 a.m. About midnight the father, who slept with the boy, observed him to be breathing rapidly, and was particularly alarmed at the loudness of the heart-sounds. He got up and took the boy out of bed, and was still more alarmed to find that the child could not stand. Having been thus aroused, the child became violently delirious, striking out with his fists, kicking, and biting on the slightest interference with his person. On being put back to bed, he could scarcely be kept under the blankets on account of the violence of his movements. The father did his best to keep the boy quiet until the mother's return. When she saw the state of matters her suspicion of poisoning was aroused, and on examining the bottle from which she had given the dose, she found it to be labelled, "Poison." She, therefore, immediately sent for medical aid. When I arrived I found the boy in the state described above. When left alone in bed, he came out from under the blankets, and wandered on hands and knees aimlessly about the bed, occasionally crying out. The delirium and violent movements were increased when efforts were made to control them. The movements were at no time convulsive. The face was flushed, not cyanosed. The pupils were widely dilated, and failed to respond to light. The pulse and respiration were strong and rapid, and the heart sounds were very loud. The boy was unable to speak. I found the suspected bottle to contain glycerine of belladonna. I ascertained that the boy had not vomited. As the evidences of belladonna poisoning were undoubted, I washed out the stomach with the syphon. A good deal of the water first introduced was vomited over the child's nightshirt, which was of a dark brown colour, so that it was difficult to tell from its appearance whether it contained belladonna extract or not. That which came through the syphon was clear. There was no odour of belladonna from the washings. Considering that five hours had elapsed since the poison was taken, I deemed it highly improbable that any of it still remained in the stomach.

After completing the washing of the stomach, I gave a hypodermic injection of a quarter of a grain of morphia sulphate, and a tablespoonful of castor-oil, by the mouth, which was swallowed. In a short time the boy became quiet, and I left the house, having directed the parents to send for me should any further alarming symptoms arise.

I was unable to see the patient again until 11 a.m. Then I ascertained that he had slept well till ten, and on awaking still exhibited symptoms of the same kind, but less aggravated. The delirium now was of a hilarious character, somewhat resembling drunkenness. The child laughed and chatted about his school experiences, games, fights, etc. The bowels had not yet moved. I then gave another hypodermic injection of one-quarter of a grain of morphia sulphate and three grains of calomel by the mouth. The boy did not again sleep till about eleven o'clock. During the whole of that day somebody had to remain constantly at the bedside to keep him in bed. Several times he attempted to bite those trying to control his movements. Although the mouth was very dry and the tongue was constantly rolled about, the boy refused food or drink, and spat out whatever was introduced into his mouth. At two in the afternoon, the bowels not having yet moved, the mother gave a small soap-and-water enema, after which there were three or four liquid motions. The boy continued in the same mental state till about 11 p.m., when he fell asleep. Shortly after 11 p.m. I visted the patient, and finding him sleeping tranquilly, I judged it best to do nothing. He had a good night's rest. The following morning at ten I found him awake, and presenting no abnormal

[1] *Lancet*, 1901, 1, p. 1198.

symptoms, except dilatation of the pupils, which persisted for four days. No rash at any time appeared upon the body, and there was no peeling of the skin. After recovery the boy seemed to have no recollection of the incidents which occurred during his illness.

For other cases of poisoning, *vide B.M.J.*, 1899, 2, p. 1792, where its application to a woman's breast caused toxic symptoms; *B.M.J.*, 1904, 1, p. 189, where symptoms arose with the tenth drop in the eye of an atropine solution, strength one grain in two drachms; *B.M.J.*, 1897, 1, p.1157, a case of recovery after three grains of atropine on treatment with morphia.

A boy, *æt.* 12, ate some ripe belladonna fruits whilst on a country excursion. On returning hime he went to bed as usual, but awoke at 2 a.m. He was then delirious, and had such violent convulsions that he was unable to sit still. He talked incoherently. He apparently could not swallow, as he put water into his mouth and spat it out again, the effort throwing him into severe convulsions. At 10 a.m. the delirium and convulsions still continued; there was a marked flushing on the legs and face, and the latter was somewhat swollen. The throat was red and he showed aversion to water. The pupils were widely dilated. The dilirium continued, with convulsions, all day and during the next night, though occasionally he spoke rationally. He was flushed, and appeared like a child in the early stage of a scarlatina rash. At noon—about forty-two hours after (as was supposed) the fruits were eaten—one-third of a grain of a salt of morphine was injected hypodermically. The boy slept, and next day awoke well.

The following case of poisoning due to one application of linimentum belladonnæ to the lumbar region of a healthy male seems worth recording. The patient, a strongly-built male of about 30 years, applied liniment of belladonna to his back at 9 p.m. An hour later he seemed to be quite off his head, and remained so during the night, getting no sleep whatever. The following morning he was found lying in bed with a startled, hunted expression upon his face, eyes bright and shining, pupils widely dilated, mumbling and muttering speech, dry tongue and skin, trembling lips and hands, delirious pulse 120 compressible and running. He vomited several times. After bowels and kidneys were made to act well and opium gave him some sleep he began to improve. Hypodermic injections of pilocarpin nitras had an excellent effect in quieting the pulse and making him more comfortable. That night the head symptoms were much better and in two days he was practically well. The amount of liniment used was about three drachms. The skin over the back was sound and normal in appearance.[1]

A boy, aged four years and three months, suffering from whooping-cough, was given two minims of tincture of belladonna and two grains of bromide of potassium, to be taken every four hours. Within half an hour of the administration of the first dose, the mother told me, a red flush came out round the child's neck and on his chest. The mouth seemed dried up and the child was slightly delirious, but in about an hour he got much better. A second dose of the mixture was given four hours after the first, and again in half an hour the child suffered in the same way and was violently sick. On arrival of the doctor the child was found almost covered with a scarlatiniform rash, chiefly on the neck and chest; the pulse was rapid (120) and very feeble; the mouth was dry; the pupils were fully dilated, a narrow ring of the iris only being visible; and the temperature was slightly raised (100° F.). The child was only semi-conscious and was with difficulty roused and could not speak. On administering brandy consciousness and speech returned, the pulse slowed, and the pupils began to contract. The next morning he had quite recovered, except for a very faint rash which entirely disappeared within twenty-four hours.

In view of the very small dose administered this idiosyncrasy is worth recording.

There appears to be some uncertainty of opinion as to whether belladonna poisoning can follow eating the flesh of a rabbit which has been feeding on *Atropa belladonna*. Blyth says[2] "It is the general opinion that rabbits may eat sufficient of the belladonna plant to render the flesh poisonous and yet the animals themselves may show no disturbance in health. But this must not be considered adequately

[1] *B.M.J.*, April 25th, 1908, p. 987.
[2] Blyth, A. W. and M. W., "Poisons, their Effects and Detection," 1920, p. 394.

established." The incident related below seems to prove that the general opinion referred to by Blyth is correct.

A greengrocer, while buying vegetables for his shop, was given a rabbit which had been snared that morning in a district where *Atropa belladonna* is known to grow. Having previously been a fishmonger and poultry-dealer, he prepared the rabbit himself for consumption, noting that it appeared to be in every way in excellent condition. The rabbit was stewed by his wife, and at 1 p.m. these two and their assistant, a girl aged 20, partook of the dish. After the meal, the girl, while washing up the plates, noticed her mouth was dry and procured a drink of water, being shortly joined by the elder woman in the same plight. Soon afterwards they were greatly alarmed by "failing vision," and on calling to the man for assistance, found him in a similar condition. One of us (J. R. B.) was then summoned and saw the patients about 3 p.m.; all presented the typical picture of mild belladonna poisoning—pupils dilated, dry mouth, giddiness and rapid pulse. After the usual procedure in cases of poisoning, inquiries were made for the source of the poison, but the only thing taken in common was the rabbit for dinner. At 9.30 the same evening we saw the cases together. The man and his wife were better, though the pupils were still widely dilated, and a marked scarlatiniform rash had appeared on the girl. We elicited the information that a dog and a cat in the house had been given some of the rabbit, and an examination of the pupils of both showed them to be dilated and immobile to light. A portion of the rabbit, including the liver, was taken and submitted to analysis. The report on the analysis states that chemical examination of the portion of cooked rabbit showed that a small quantity of alkaloid was present, giving a definite reaction by the Vitali method for belladonna alkaloids.[1]

Poisoning by Datura stramonium

Source and Method of Occurrence. The plant is known as thornapple. It is not a very common plant in England, but when once it has established itself as a weed in a garden, it is difficult to eradicate it. The flowers are attractive.

All parts of this plant are poisonous: but the *seeds* and *fruit* are considered to be the most noxious. They contain 0·2 to 0·4 per cent. hyoscyamine.

Stramonium cigarettes made from the leaves are smoked to relieve an attack of asthma.

One of the methods of poisoning adopted in the East, not so much with the intention of destroying life as of facilitating the perpetration of robbery, consists in administering to persons either the seeds mixed with dates or figs, or in a powdered state, or by mixing a strong decoction of them, in curry, or in some other highly flavoured article of food. Chevers has given a very complete account of the Hindoo system of poisoning by *dhatoora*.[2] It appears that the *Datura fastuosa*, *alba*, and *stramonium* are the principal sources of the poison in India. The Thugs employ this poison with the object of rendering their intended victims helpless. Delirium and insensibility soon follow, and sometimes death is the result. Occasionally there are considerable outbreaks of accidental cases[3] as well as numerous cases in young children from eating the seeds of the thornapple.[4]

Toxicity and Fatal Dose. The active principle, a mixture of hyoscine, atropine and hyoscyamine, is extremely toxic, and as the plant contains

[1] Firth and Bentley, *Lancet*, October 29th, 1921.
[2] "Med. Jur. for India," 1856, pp. 121, 549, 591. See also *B.M.J.*, 1892, 2, p. 641.
[3] Anderson, T. F., *et alia*. *E. African Med. Jour.*, 21 : 355 (1944).
[4] Hughes, J. W., and Clark, J. A., *J. A.M. A.*, 112 : 2500 (1939).

approximately, ½ to 1 per cent. of alkaloids, it must be considered dangerous. The seeds are highly poisonous, inasmuch as they contain a larger proportion of alkaloids than other parts of the plant. Death may take place although the whole of the seeds are ejected. A child, *æt.* 2, swallowed about 100 seeds of stramonium weighing sixteen grains. The usual symptoms were manifested in an hour, and the child died in twenty-four hours although twenty seeds had been ejected by vomiting and eighty by purging.[1] Sufficient alkaloid to destroy life had been absorbed from the entire seeds and carried into the blood. In a case which became the subject of a trial at Osnabrück, a woman administered to her mother a decoction of the bruised *seeds* of the thornapple, of which it was supposed there were about 125. She very soon became delirious, threw her arms about, and spoke incoherently; she died in seven hours.

Duration. The symptoms commonly come on in about ten minutes after the poison has been taken; they may be delayed from half to one hour, depending on the skill of the administrator. In accidental cases the shorter limit is that usually observed. In fatal cases death usually occurs within three or four hours; in non-fatal cases recovery takes a day or two, the effect on the pupils being the last to disappear.

Symptoms. The drug has a bitter taste, which it generally imparts to the food with which it is mixed; this is sometimes recognised when it is eaten. Following the taste there soon appear symptoms identical with those of belladonna poisoning. There is at first a dryness in the throat, attended with a feeling of faintness, headache, and giddiness, the person has difficulty in walking straight and appears as if intoxicated, while at the same time he is very restless. The pupils are dilated, he will sometimes complain of indistinctness of vision, or drowsiness, and he almost always falls asleep. The sleep may either increase to complete insensibility with dilated pupils, a flushed face, and muttering delirium, or the patient may awake and then become delirious. The delirium is characterised by great restlessness, the person affected frequently moving about, and there is a tendency to strip naked and to pick at various objects. There is great thirst. After a time the patient becomes again insensible, and is greatly exhausted; sometimes convulsions occur, with low muttering delirium, and at length he dies. If, as more frequently happens, he recovers, the insensibility persists for a day or more, and the patient remains occasionally in an idiotic state, able to speak, but not to understand for some time longer, and he has no recollection of what has occurred after the poisonous meal. Sometimes vomiting is an early symptom, although this is rare.

Treatment. Cases should be treated on the same lines as cases of belladonna poisoning (*q.v.*, p. 656). The bowel should be washed out frequently to get rid of remnants of crushed seeds.

Post-mortem Appearances. There is, beyond finding bits of the plant or seeds in the stomach or intestines, nothing to indicate the cause of death.

Analysis. The *seeds* of stramonium, from which accidents have most frequently occurred, are flattened, kidney-shaped, but half oval, rough,

[1] *Med. Gaz.*, vol. 15, p. 194

and of a dark-brown or black colour. The seeds are liable to be mistaken for those of capsicum. The datura seeds, however, present dots on their exterior, which on a microscopical examination are seen to be composed of convoluted ridges surrounding spaces. On the capsicum seed these convoluted ridges run nearly parallel to each other, and are joined at right angles by shorter ridges so that most of the spaces are of an oblong form, and are as lines curving round the seed : but in datura, the ridges are more convoluted and irregular, joining at acute angles and circumscribing irregular spaces. Of the dry *Datura stramonium*, about eight seeds weigh one grain. The seeds of the *Datura fastuosa* are very similar in size and general appearance. The seeds of *Datura alba* are larger, flatter, and much lighter-coloured, but have similar microscopical characters.

The *leaves* of the common *Datura stramonium* are well characterised by their peculiar shape. For the reactions of the alkaloid, *vide* Atropine pp. 657 *et seq.*

At noon, Mrs. H., aged fifty-three, took a teaspoonful of an anti-asthmatical powder in mistake for liquorice powder. At 12.45 p.m., when sitting down to dinner, she could not understand a dry feeling in her mouth, which sipping water did not affect in the least ; at the same time her sight became blurred, and she experienced a peculiar sensation of swelling in her eyes ; lifting the glass to moisten her lips she exclaimed at the weight of it, and feeling herself getting worse she told her husband to go for the doctor, and from this time remembered nothing till about 6 or 7 p.m. When I saw her at 2 p.m. her condition bore a remarkable resemblance to a case of *delirium tremens* ; her face, however, which was markedly pale, was not expressive of suspicion not anxiety, nor was there any clammy perspiration, the skin being perfectly dry. Her eyes were bright and staring, the pupils dilated but not excessively, and absolutely insensible to light. The flow of ideas was very rapid, and her speech so fast that only at times could any sense be made of what she said. Mirthful delirium and hallucinations were very prominent, but illusions and delusions were markedly absent. Although she often attempted to rise she seemed unable to do so from inco-ordination of the lower extremities. Sensation, however, was perfect. The power of swallowing at first seemed absent, but if she was prevented from returning what was placed in her mouth she swallowed it. Breathing was quiet, but the pulse was very rapid, thready and compressible. After using the stomach-pump freely nitrite of amyl and digitalin (hypodermic) were administered, and shortly afterwards pilocarpin (one-third of a grain). Recovery after the use of the latter was remarkably rapid.[1]

Turner[2] describes five cases of poisoning by these seeds in children under ten years of age. They had eaten them in the scarcely ripe state, when they are not very bitter. In one hour and a half two of the children were found to be fully under the influence of the poison. They were lying on their backs, the eyes bright, and the pupils widely dilated and insensible to light ; the conjunctivæ were injected, the face deeply suffused and of a dark crimson colour ; there was difficulty of breathing, inability to articulate, and a state of complete insensibility, broken occasionally by a paroxysm, during which they would utter some indistinct sounds and throw their hands about, as if trying to ward off some threatening evil. Then they fell into a comatose state, but were easily roused into a state of violent excitement ; they grasped at imaginary objects ; there was picking at the bed clothes, with paroxysms of excessive laughter. They had no proper control over their limbs, walked with a staggering gait, and fell to the ground as if intoxicated, or in a state of complete exhaustion. They recovered under treatment in about twenty-four hours.[3]

A girl, *æt.* 5, ate about half a dozen stramonium seeds, together with some of the pulpy matrix of the fruit. Symptoms came on within twenty minutes, beginning with dryness and burning of the throat, thirst, inability to swallow, nausea and

[1] *B.M.J.*, 1898, 1, p. 1071.
[2] *Amer. Jour. Med. Sci.*, April, 1864, p. 552.
[3] See also other cases in the same journal, January 7th, 1862, p. 54.

retching but no vomiting, pain in the stomach, flushed face, giddiness, and singing in the ears. Twitchings of the muscles of the forearm were next observed and delirium set in. When seen an hour and a half after eating the seeds, the girl was lying on her back, delirious, apparently unconscious, and in convulsions. She caught at imaginary objects in the air. The eyes were bright and glistening, the conjunctivæ red and injected, and the pupils widely dilated. The delirium was of a busy kind—fits of laughter alternating with fits of crying and horror. The convulsions were general. The skin was hot, dry, and of a deep scarlet hue : the temperature normal ; the pulse small and rapid ; the respiration interrupted, but not rapid. She recovered. From six ounces of the urine voided five hours after the seeds were eaten, a substance was obtained by Stas's process which dilated the pupil when applied to the eye of a man ; and this dilatation lasted for some hours.[1]

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Poisoning by Hyoscyamus niger (Henbane)

Source and Method of Occurrence. The whole plant is poisonous, but it is chiefly from the seeds that cases have arisen, and from the alkaloids. The plant is moderately common in England, but is unattractive except by its curious lurid flowers.

In 1892, several persons suffered in a slight degree from the ordinary symptoms of poisoning by henbane after partaking of soup flavoured with the seeds, which were sold as those of celery. Two teaspoonfuls of the seeds, weighing about seventy-two grains, were put into the soup.[2]

The fumes evolved from the burning seeds, when inhaled, are a popular remedy for toothache.

The poisonous properties of henbane are due to the presence of the same alkaloids as are found in belladonna and datura, namely, *atropine*, *hyoscyamine* and *hyoscine* or *scopolamine*, therefore the symptoms and treatment are the same.

Post-mortem Appearances. There is nothing to be looked for except portions of the plant or seeds which may be identified by their botanical characters. The seeds are very small and hard ; they are pitted on the surface, and may be easily confounded with those of belladonna. They are of an oblong, oval, or pyriform shape. The leaves are peculiar in shape and other characters, by which they may be easily identified.

Analysis. See Atropine.

Cases. In *Rex* v. *Crippen*,[3] Wilcox isolated two-fifths of a grain of hyoscine hydrobromide from portions of the viscera of the victim about five months after the murder. A case of non-fatal poisoning from one-twenty-fifth of a grain hydrobromide of hyoscine has been recorded.[4]

[1] Canada *Lancet*, vol. 14, p. 161.
[2] *B.M.J.*, 1892, 1, p. 1075.
[3] C. C. C., 1910 ; see *B.M.J.*, October 29th, 1910.
[4] *B.M.J.*, 1 : 336 (1896).

"M. L., aged fifty, suffered from a severe form of cyclitis implicating both eyes and extending to the choroid and retina. Adhesions had already formed when the case came under observation. The inflammation lasted four months. Atropine, homatropine and cocaine, and atropine alternating with eserine were used with little or no effect. Six drops of a 1 per cent. solution of the sulphate of atropine were used three times a day for a considerable time with little effect on the pupil, and no perceptible effect on the general system except a slight dryness of the throat.

"As a greater effect was desired a 1 per cent. solution of hyoscine hydrobromide was tried, two drops of the solution being placed in each eye.

"Five minutes afterwards she complained of giddiness and a feeling of lightness in the head; she staggered, and had to be assisted to bed. Then great dryness of the mouth and throat with thirst supervened. The giddiness increased, gradually the senses became confused, and the power of speech was lost. Complete muscular relaxation became pronounced, and she became rapidly unconscious. The breathing was slow, and occasionally deep and sighing. The face was flushed, the pulse full and regular.

"This unconscious stage lasted for about four hours and was succeeded by a period of semi-consciousness. At times the patient seemed to have perfectly recovered; at other times she was quite delirious. She talked incessantly, was occasionally irritable, although on the whole it took the form of a pleasant delirium. She recalled funny incidents long since past, made jokes about everybody and everything, and kept those round about her, but for their anxiety, greatly amused with her witticisms.

"After about two hours of this delirium, she became gradually calmer, and then dropped off into a sound and seemingly natural sleep, which lasted for about an hour and a half. When she awoke, she remarked that she had not slept so well for a long time. She had no remembrance of the events of the night further than being assisted to bed. There were no evil after-effects, and by the afternoon, when I saw her, she seemed in her usual health."

The following case,[1] is instructive :—

"On October 30th, 1903, a man, aged sixty-nine years, came to me complaining of cramps in the muscles of the legs at night and also of senile tremor of the hands. He had been under my care at intervals for several years. Five years ago I attended him through a severe attack of basic pneumonia, which was accompanied by much excitement, and then one-hundredth of a grain of hyoscine hydrobromide was used hypodermically with great benefit. At the present time he was in fair health for his age. The pulse was rather quick—about ninety per minute. The heart sounds were normal. The urine was highly coloured: it was free from albumen and sugar. I ordered him one two-hundredth of a grain of hyoscine hydrobromide in one drachm of water to be taken at bedtime. At 9.45 p.m. on the same day I was hurriedly summoned to see him. I was informed that at 9.15 p.m. one teaspoonful of the medicine was given him by his son (this was confirmed by the amount absent from the bottle). After swallowing it he undressed and got into bed. While undressing he remarked to his wife that his throat felt very dry, and she noticed that his speech was rather thick. Two or three minutes after he got into bed his wife heard him breathing very deeply; she tried to rouse him, but could not, and sent for me. I found him half an hour after he had taken the medicine deeply comatose with stertorous breathing and flushed face. His pulse was eighty per minute and regular, his pupils were dilated and equal, and the conjunctival reflex was very slight; I could not rouse him by any means. I injected one-tenth of a grain of strychnine hypodermically and gave one ounce of brandy by the rectum. At 10.45 p.m. there was no improvement; the coma, if anything, was deeper, the conjunctival reflex was quite absent, the pulse was smaller and weaker, and there was some twitching of the arms and legs. I injected one-sixth of a grain of morphine with one grain of caffeine citrate. In a few minutes the pupils became less dilated, but otherwise there was no change. At 11.45 p.m., I washed out the stomach, and passed into the stomach about eight ounces of strong black coffee and one ounce of brandy. At 1 a.m. the conjunctival reflex began to return, and the patient flinched on pinching the skin on the inner side of the arm. Improvement slowly set in, the breathing became less noisy, and

[1] *Lancet*, 1 : 24 (1904).

the pulse fuller and stronger. At 3 a.m. he could be partially roused for a moment by pinching and shouting, but at once relapsed into a somnolent condition; the pupils now reacted to light. At 5 a.m. he made an attempt to speak and swallowed some coffee. At 8 a.m.—that is, eleven hours after taking the dose—he was really conscious and able to speak for the first time, and he thenceforth made an uneventful recovery.

"The prescription which was for one two-hundredth of a grain of hyoscine hydrobromide in one drachm of water, with orders to send two ounces, was dispensed at a very trustworthy druggist's, and a very unfortunate and dangerous, though instructive, mistake was made in dispensing it. The druggists in question gave me, I am glad to say, every facility for investigating the matter. It appears that they had two establishments. At the one to which the prescription was taken they had no hyoscine in stock, so the dispenser sent a written message to the other place for 0·80 gr. of hyoscine hydrobromide in two ounces of water, which is equal to sixteen two-hundredths of a grain, the exact amount he required for the whole bottle of medicine. The dispenser at the second shop seeing decimals were used, took it for granted that it was a foreign prescription, and that 'gr.' stood for grammes, and sent 0·80 gramme, which the first man sent to my patient. The result of this was that he got more than fifteen times as much as was intended, or about one-thirteenth of a grain. This appears to be a good object-lesson in the danger of the present condition of weights and measures in the Pharmacopœia, where it is now optional for either the decimal system or the apothecaries' measure to be used. Surely the time has come for the rational and scientific metric system to be enforced as the official standard."

Poisoning by Cannabis Indica

Source and Method of Occurrence. The dried flowering or fruiting tops of the female plant, grown in India, used to be the source of two official pharmacopœial preparations but are now omitted from the pharmacopœia.[1] Preparations of the plant form the basis of the well-known bhang or hashish used by millions in Eastern countries as a means of intoxication, and as a result, poisoning by the drug in both acute and chronic forms is common. *Bhang* consists of the dried leaves of the Cannabis sativa. *Ganga* consists of the dried flowering tops of the female plant, specially grown so that there is a large amount of resinous exudate and as little leaf as possible.

Charas is the resinous exudate of the plant and the most powerful of the three varieties.

Hashish, as sold in Egypt, roughly corresponds to the Indian charas, but contains more vegetable and mineral matter.

On fractional distillation under reduced pressure of the alcoholic ether or petrol ether extract, several products are obtained, including a reddish oil which solidifies below 60° C., and which is known as Cannabinol. This is probably a mixture of two or more active principles. More recent work[2] on the separation of pure cannabinol and the fractionation of the remainder of the crude cannabis extract indicates that though cannabinol is a powerful poison, it is not the only toxic substance present. Fractions free from cannabinol were found to cause deep sleep and, in large doses, death. Cannabinol itself does not produce the Gayer effect (anæsthesia of the cornea) which is given by crude extracts.

In England poisoning by Cannabis indica is rare, and only occurs as the result of accidental overdoses. There is, however, a certain amount of evidence to show that it is used as a narcotic for self-administration in some towns.

[1] Extract of Cannabis (dose 15 mg.) and Fluid extract of Cannabis (dose 0·1 cc.) are official U.S.P. drugs.

[2] Work, Bergel and Todd. *Biochem. J.*, 1939, *33*, 123.

Toxicity and Fatal Dose. The fatal dose of the drug or its preparations is unknown, but doubtless habit has a good deal to do with it, as in the case of opium, in countries where the drug is habitually used.

Duration. In acute cases toxic symptoms appear within a quarter to half an hour of taking the drug. Death has followed in twelve hours, but as a general rule the patient recovers even after exhibiting severe symptoms of poisoning.

Symptoms. These are somewhat similar to those of alcohol. They consist at first of a pleasurable excitement of the mind; giddiness and sleepiness soon supervene, followed in fatal cases by coma and collapse. The following is a fairly typical case :—

The patient, a boy, *æt.* 12, took a dose of the medicine (ten minims of the tincture) and in a few minutes said he felt a burning pain in the pit of his stomach and soon became strange in his manner, saying that his legs were jumping about and that he heard a ticking like a watch, that he saw the room on fire and the pictures falling down, etc. On admission to the hospital he looked extremely ill, very pale, anxious and distressed. He did not complain of pain, but was much collapsed. His pulse was 120, being scarcely perceptible at the wrist; the pupils were dilated, but acted sluggishly to light. He was at once laid on a couch, was covered with blankets, and was given two drachms of brandy in hot water, and immediately after this ten grains of citric acid in syrup of lemon. A blister was applied to the nape of the neck. He soon began to revive, his colour improved, and in about two and a half hours he had recovered and said that he felt quite well—his headache had gone and he was able to walk back to his home.[1]

Or the following, from six grains of the extract :—

A few weeks ago I was called about 11 p.m. to see a young woman who had, it was alleged, "gone out of her mind." I thought at first I had to do with an ordinary hysterical fit, but closer observation convinced me that although the symptoms were a curious caricature of some hysterical attacks there were other symptoms that made such a diagnosis untenable. Fits of laughter and incoherent ravings alternated with comparatively lucid intervals. She complained of various hallucinations and delusions, chief of which were a complete perversion of the relations and lapse of time and a loss of identity. She was now herself, now again a different individual, and the modifications of her behaviour in relation to her dual personality were grotesque. I was entirely at a loss for the cause of the patient's condition until her mother volunteered that she had been taking Indian hemp. Like many other foolish people they had purchased a book on family medicine. The girl had suffered severely from headache for some time, and after consulting the book they pitched upon Indian hemp as the likely remedy. A local chemist made them a dozen pills, each containing one-half grain of the extract. The girl took one with no relief, then two with a similar effect. She then concluded that they were "a fraud," and to demonstrate it swallowed the remaining nine with the result that I found her four hours after in the above-mentioned condition. I administered strong coffee and gave her a few doses of strychnine, and she quite recovered on the following day.[2]

The two following cases are of interest from the smallness of the doses that caused the symptoms, seven minims, and twenty-four minims in two separate doses respectively :—

A lady, *æt.* 30, suffered from symptoms of poisoning, following a dose of only seven minims of the ordinary tincture. After taking this quantity in a mixture she became drowsy, her vision was dimmed, she was sick, had great thirst with dryness of the fauces, and slept heavily. Four hours after she had taken the tincture she was still in a state of narcotism, very drowsy and not easily roused. The pupils were fully dilated, the eyes suffused, the tongue dry, and the pulse

[1] *Lancet*, 1896, 2, 1078.
[2] Baxter-Tyrie, *Lancet*, 1897, 2, p. 1452.

small and quick. The symptoms were followed by profuse perspiration. The next day the symptoms had disappeared with the exception of some dryness of the mouth and feverishness.[1]

In June, 1897, I saw a patient, aged thirty-three years, who complained much of an attack of migraine. I ordered her a mixture containing ten grains of strontium bromide and twelve minims of the tincture of Indian hemp in each dose, one to be taken every fourth hour till the pain was relieved or until four doses had been taken. After the first dose she felt much better and went out to make some purchases. On returning, about four hours after taking the first dose, she took a second dose; within a few minutes she felt a strange feeling and an inclination to jump down the stairs. This she knew was absurd, so she clung to the banister; she then went into her room and to bed, when I was sent for. I found her in bed in an excited, joyous state, quite conscious, but, as she said herself, unable to control herself: she talked incessantly and assured me that she had such a good story to tell me, etc. Her eyes were brilliant and the pupils were slightly dilated, but they reacted to light: her pulse was full and soft. She mentioned that she had a creeping sensation in her arms and legs. As it was more than an hour since she had swallowed the medicine and the symptoms were not urgent I did not administer an emetic, but gave her some strong black coffee, and assured her that she would be quite well in a short time. In about half an hour she became quiet, and she then slept for an hour, on awaking being quite herself.[2]

Treatment. The same as for opium.

Post-mortem Appearances. Nothing in any way characteristic.

Beam's Test. A petroleum ether extract of hashish (or gastric contents containing hashish) evaporated to dryness gives a purple colour when a drop of alcoholic potash is added. If the residue from the petrol ether is oily a colour may fail to appear until more alcoholic potash is added and the mixture is gently warmed.

This test is not obtained with tincture of Cannabis indica. Beam altered his test so that a reaction could be obtained from all kinds of cannabis preparations as follows :—

The petroleum ether extract is evaporated very nearly to dryness in a short test tube. To the residue is added a few cubic centimetres of a reagent prepared by passing dry hydrogen chloride gas through absolute alcohol to saturation. In the presence of cannabis extract the liquid becomes cherry red. The colour disappears on dilution with alcohol or water. Smith, however, states that even in this form the test may fail to give conclusive results ("Forensic Medicine," 8th Edition, p. 621).

Negm's Test.[3] According to Bamford,[4] many samples of cannabis which fail to respond to Beam's test give positive results with Negm's test. The residue from evaporation of a petroleum ether extract of hashish is mixed with 2 ml. of Negm's reagent (vanillin 0·4 g., acetaldehyde 4 drops, 95 per cent. alcohol 20 ml.) and 2 ml. of concentrated hydrochloric acid. A positive result consists in the appearance of a pale green colour which quickly turns a slate grey and, in about ten minutes, indigo-blue. Still later it changes to violet which is intense after about an hour.

Microscopic examination of the crude drug shows numerous glands in the tissue of the leaves and numbers of pointed glandular hairs.

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[1] *Lancet*, 1871, 2, 493.
[2] Roche, *Lancet*, 1898, 2, p. 1701.
[3] Thesis, Strasbourg, 1938.
[4] "Poisons," 2nd Edition, Churchill, 1947, p. 291.

A. Deakin, " Death from taking *Cannabis indica*." *Ind. Med. Gaz.*, 1880, 15, 71. (*Note.* This seems to be the only fatal case recorded as directly due to a poisonous dose, but deaths from its habitual use occur occasionally in the East.)
Hooper, *Pharmac. Journ.*, September 19th, 1908.
Beam, " Wellcome Tropical Research Lab.," 4th Report, Vol. B., 1911, p. 25.
Bromberg, " Marihuana Intoxication." *Amer. J. Psychia.* 1934, *91*, 303; *S. Amer. Med. Assoc.* 1939, *113*, 4.

Poisoning by Capsicum minimum (Cayenne Pepper)

In small quantities this is a well-known stimulant, and is in common use as a condiment. It has a hot, fiery taste, which lasts for a long time. It is a powerful stimulant, and in large doses produces a feeling of heat in the throat, difficulty of swallowing, pain in the stomach, and inflammation of the gullet and stomach. Locally applied it causes redness and even blistering of the skin. The toxicity of capsicum is low[1] and there is no undoubted instance recorded of its having proved fatal.

Guinea Pepper, known as grains of paradise, is popularly considered to be highly noxious ; but there are no facts to justify this view. This kind of pepper is an aromatic condiment.[2]

Poisoning by Chailletia toxicaria

The following case, reported by Dr. Renner, *B.M.J.*, 1904, p. 1314, is inserted for corroboration by subsequent cases that may occur.

J., a Mendi labourer, aged about twenty-four years, was admitted on the morning of November 18th, 1903, at 10 a.m. He could not speak English fluently, and the following history was obtained from his friends.

On the morning of November 18th, 1903, some fish, on which had been sprinkled the powdered fruit of the ratsbane for the purpose of killing rats, having been given to him to throw away, he ate the largest portion of it. In about half an hour afterwards he had vomiting followed by looseness of the bowels and general trembling. On his admission into hospital about 10 a.m. he again vomited, and his condition then, as described by himself through an interpreter, was as follows : " He was feeling very weak and unable to walk, his legs were dead, he was losing power over his arm, and he was feeling very bad." On examination in the ward it was found that the patient was suffering from paralysis of the lower extremities. The tendon reflexes were abolished. There was marked hyperæsthesia of the inner side of the thigh and legs. Firm pressure of the muscles of the calves gave unusually severe pain. The action of the bladder and rectum remained undisturbed. Although the pupils were normal yet the patient's vision was not acute. There was some want of co-ordinating power in the muscles of the upper extremities. The power of deglutition was not affected. The patient's condition was stationary for about a fortnight, when signs of improvement began, and this continued gradually until January 22nd, 1904, when he was discharged with only a slight inability to walk.

The case is unique, as it is, I believe, the first overt and obvious instance of poison by ratsbane recorded in man. The symptoms observed in this case have been frequently observed in sudden cases of a disease characterised by paralysis of the lower extremities in young persons of both sexes and of various ages, but especially in those between the ages of twenty and forty.

The origin of this disease has been veiled in great obscurity ; even experienced medical men have been baffled in their efforts to discover it. The disease has generally been regarded as mysterious by the people, because the country doctors, who have the true knowledge of the cause of the disease, keep it a secret, attributing it to some mysterious influence of the devil, or to witchcraft, or something occult, and thereby make much profit from their knowledge, and wield great power over the masses.

There is now no doubt in my mind that these cases of sudden illness resulting in paralysis are due to poison by the ratsbane, and the numerous deaths commonly

[1] Carmichael, E. B., *Jour. Pharm. & Exp. Therap.*, 66 : 6 (1939).
[2] Snape, " Poisoning by Cayenne Pepper," *Med. Times*, Lond., 1849, 19, 552.

reported to be from poison may also be attributed to the effective use of this substance. The symptoms observed in the domestic animals when under the influence of this poison are the same as in man. Cats and dogs which may happen to be poisoned by eating fish sprinkled with the powdered fruit show in a few minutes great distress, they vomit, rush about frantically, and their legs soon become paralysed. They then lie down helplessly, breathing quickly, the forearm twitching and quivering; they ultimately die, apparently from paralysis of the respiratory muscles.

This poison is derived from the fruit of the *Chailletia toxicaria*, natural order Chailletiaceæ. It grows in West Africa and South America. In Sierra Leone it is commonly called "broke back" from its effect in producing paralysis of the lower limbs. In the hinterland it is known by the Mendis as "magbevi," and by the Timnes as "manuch."

Poisoning by Chelidonium majus (Greater Celandine)

Source and Method of Occurrence. The plant is a fairly common English wild flower belonging to the natural order Papaveraceæ. It occurs in nearly every county of the British Isles. It is said to contain two chief alkaloids chelidonine which resembles morphine and sanguinarine which causes salivation, excitement and tetanus.

The juice acts as an irritant externally, and internally causes nausea, vomiting and diarrhœa. It has caused poisoning in cattle but there are no records of its use in human beings.

Poisoning by Cicuta virosa (Water Hemlock)

Source and Method of Occurrence. The water hemlock, or cowbane, has given rise to several fatal accidents, its roots having been mistaken for parsnips. The whole of the plant is poisonous; but the roots are the most active, especially when gathered early or late in the year. It contains an active principle known as cicutoxin, which acts like picrotoxin, causing medullary convulsions. Holmes[1] states that the American species Cicuta maculata contains a volatile alkaloid similar to coniine in its seeds.

Symptoms and Effects. The symptoms produced by eating the roots are pain in the stomach, vomiting and giddiness, severe and continued convulsions, followed by paralysis. A man ate a portion of the root of this plant in a cooked state. It had a sweetish taste, and was of the colour of a parsnip. Half an hour later he felt giddiness, and dryness of the throat. He walked home with difficulty. In about an hour and a half the legs were paralysed, the arms benumbed, and their movements weak: the skin was warm and dry—the pulse 90. An emetic was given. Within two hours he was able to stand, and with difficulty walked across the room. He passed much urine, and had hallucinations. Within seven hours the legs were cold, pupils dilated, skin and throat dry, with occasional delirium. There was no purging. Two days later he recovered.[2]

Egdahl[3] has collected the history of 46 cases of cicuta poisoning, twenty-one of which were fatal. Nausea, vomiting and convulsions were the outstanding symptoms.

[1] *Pharm. Jour.*, 1911, p. 430.
[2] *Lancet*, 1871, 2, p. 396.
[3] Egdahl, A., *Arch. Int. Med.*, 7 : 348 (1911).

Gomportz[1] reports similar cases of poisoning by the American variety of water hemlock (*C. maculata*). All those who ate the root had nausea followed by convulsions. The convulsions were tonic and clonic. The pupils were widely dilated, the jaws were clenched with twitching of the facial muscles and frothing at the mouth. All of the cases recovered.

In addition to stomach lavage intravenous barbiturate therapy may be adopted.[2]

Analysis. Detection must depend, since there are no reliable chemical tests for cicutoxin, on (*a*) botanical characteristics of fragments found in the stomach, and (*b*) pharmacological action of an ether extract of the gastric and intestinal contents—convulsions of medullary origin in the frog.

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Pohl. "Zur Kenntn. d. gift. Bestandtheil d. Œnanthe Crocata und *Cicuta virosa*." *Arch. f. Exp. Path.*, 1894, 34, 259.

Poisoning by Cinnamomum camphora

Source and Method of Occurrence. Camphor is a natural product from the plant *Cinnamomum camphora*, and is also produced artificially. It has never been used for homicidal purposes, but cases of poisoning from accident and suicide are reported. A common cause of camphor poisoning is the ingestion of "camphorated oil" and of "moth balls" by children.

Toxicity and Fatal Dose. Camphor cannot be said to be a very poisonous substance. Its action is due to its stimulating effect on the midbrain and medullary centres and possibly the cord. It causes mental excitement, giddiness and inco-ordination and with large doses (40 grams) irregular cyclic convulsions resembling epilepsy. Its action is in many respects similar to that of picrotoxin. It also has a local irritant action on the bowel and also affects the kidney, causing anurea. The toxic dose varies greatly.

The smallest recorded dose in an adult that has caused alarming symptoms is twenty grains, and thirty grains have proved fatal to an infant of eighteen months. A case is recorded[3] in which twelve grains of camphor in oil caused the death of a child, *æt.* sixteen months, in seven hours.

Symptoms. The symptoms vary greatly but usually there is nausea and vomiting followed by excitement, giddiness and delirium. Unconsciousness may occur and epileptiform convulsions may last for a considerable time. Death is not common but convalescence may be very protracted.

A woman swallowed about *twenty grains* of camphor dissolved in rectified spirit of wine mixed with tincture of myrrh. In half an hour she was suddenly seized with languor, giddiness, partial loss of sight, delirium, numbness, and tingling and coldness of the extremities, so that she could hardly walk. The pulse was quick and respiration difficult, but she suffered no pain in any part. On the administration of an emetic she vomited a yellowish liquid smelling strongly of camphor. In the

[1] *Jour. Amer. Med. Assoc.*, October 16th, 1926.
[2] Miller, M. M., J.A.M.A., 101 : 852 (1933).
[3] *B.M.J.*, March 15th, 1924.

evening the symptoms were much diminished, but she had slight convulsive fits during the night. The next day she was convalescent; the difficulty of breathing, however, continued more or less for several weeks. An infant of fifteen months died from the effects of some camphorated oil given to it by mistake. Convulsions ensued, and death took place in thirteen hours. A woman gave about thirty grains (half a teaspoonful) of powdered camphor to each of her three children as a vermifuge. Two of the children were respectively of the ages of three and five years; the third was an infant aged eighteen months. The first symptoms were paleness of the face, with a fixed and stupid look. Delirium followed, with a sense of burning in the throat, great thirst, vomiting, purging, and convulsions; and in one child the convulsions were most violent. The two elder children, after suffering thus for three hours, fell into a comatose sleep, and on awaking the symptoms passed off. The infant died in seven hours, not having manifested any return of consciousness from the first occurrence of convulsions.[1]

An instance is recorded by Berkholz of recovery of a young girl who had taken fifteen grams of camphor, suspended in water. After washing out the stomach, chloral and potassium bromide were administered as antidotes, the cure being complete in a few days. Toxic symptoms in this case did not supervene until after the lapse of two hours, when intense cephalalgia, vomiting, convulsions, and coma supervened, the respiration being rapid and the pulse full. Recovery is attributed to the fact that before taking the dose the patient had partaken of a full meal, largely composed of carbohydrates, which may have formed in the intestines the non-toxic glyco-camphoric acid. The author recommends the use of large doses of sugar as an antidote.[2]

The following unusual case is reported by Dr. Wilkinson :[3]

A Chinese coolie's wife brought a little girl, aged five years, to my surgery, and gave the following history : " By mistake the child had been given half a (Chinese) teacupful of crude camphor oil instead of tea." This oil is used by the natives as a preventive for mosquito bites, and contains from 40 to 50 per cent. of crystallisable drug which precipitates after standing for several days.

The mother informed me that the accident had occurred about one hour and a half before coming to me, and that, directly after swallowing the oil, the child had fallen on the floor in convulsions, which lasted twenty minutes; the eyes were wide open, the face livid, hands clenched, feet cold, and legs outstretched. Towards the end of the fit the patient frothed at the mouth and vomited, but the ejecta were not blood-stained. After vomiting the child drank half a cupful of water, and was then brought to me. When I saw it, it had just vomited slightly for a second time; it looked pale and languid, and was apparently nauseated. The pulse was 100, the temperature not taken.

I immediately tried to promote emesis by ipecacuanha, and after pushing the drug succeeded in obtaining very free and repeated results. The vomit, beyond smelling strongly of camphor, presented no other peculiarities, and was quite free from blood.

The little patient soon fell asleep, and after a time was taken home. I was informed next day that she was running about and playing, and apparently quite well.

To say nothing of the camphor still held by the oil in solution after the precipitate had formed, and only reckoning the quantity by weight of the latter, this child must have swallowed at least from one hundred grains to two hundred grains of pure drug '

For a case with very severe collapse after eating 3 drachms of pure camphor, *vide* *B.M.J.*, 2, 1895, p. 660.

[1] *Jour. de Chim. Med.*, 1850, p. 507.
[2] *Bull. Comm.*, 27, 35, p. 1; *Pharm. Jour.*, 1899, p. 492.
[3] *B.M.J.*, 1 : 299 (1898).

Treatment. Must be conducted on general principles (*vide* pp. 250 *et seq.*). The convulsions may be controlled by one of the barbiturates.

Post-mortem Appearances. Nothing characteristic except the smell.

Analysis. There appear to be no specific tests for camphor better than its smell, taste, and familiar physical properties. (Melts at 176° C.; is spontaneously volatile: almost insoluble in water, soluble in alcohol, ether, chloroform and benzene.)

A case of poisoning by camphor would be recognized by the odour of the breath, a symptom which would attract the attention of a non-professional person. The presence of this substance in the stomach would be at once indicated by its odour. It can be separated by distillation, and extracted from the distillate by benzene.

In the body, camphor is oxidised to camphorol, which is excreted in the urine as the glycuronide.

Poisoning by Citrullus Colocynthis (Colocynth)

Colocynth fruits (bitter apples) are marketed in dried and powdered form as a purgative and as an insecticide.

Colocynth has occasionally produced death, preceded by symptoms of gastric and intestinal irritation, and excessive purging. It is occasionally used for procuring abortion. Christison recorded the death of a young woman from a teaspoonful and a half of the powdered pulp. Roques states that less than sixty grains of the powder, in decoction, has proved fatal; whilst recovery has taken place after three times that amount.[1]

Analysis. The active principles of colocynth appear to consist of an alkaloid and a glucoside (Power and Moore, *J.C.S.*, 1910, 97, 99); both are described as possessing purgative properties. Dragendorff succeeded in detecting colocynth in extracts from the bodies of poisoned cats, though Tidy failed in the case of a human being.

Colocynth extracts give a cherry red coloration (sometimes brown at first) with a freshly prepared solution of 1 or 2 mgrm. of molybdic acid in 1 cc. of pure sulphuric acid (Fröhde's reagent), and a transient blood-red colour with a 0·5 per cent. solution of ammonium vanadate in sulphuric acid.

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Tidy, "On Poisoning by *Cucumis colocynthis*." *Lancet*, 1868, 1, 158.

Poisoning by Claviceps purpurea (Ergot)

Source and Method of Occurrence. Ergot consists of the dried mycelium of Claviceps purpurea, a fungus which infests many cereals, but which principally occurs on rye. As commonly seen in bulk, it is the grain of rye spoilt and permeated through and through with the threads of the fungus.

Ergot has long enjoyed a reputation for its action upon the uterus (its action is essentially that of causing unstriped muscle to contract, and it, therefore, powerfully affects the uterus, which consists almost

[1] Husemann, "Handb. d. Toxicol.," p. 625

entirely of unstriped muscle) and hence is a favourite drug to administer for the purpose of procuring abortion, and a large number of cases of poisoning by it have given rise to criminal charges for abortion.

For its use in abortion, *vide* pp. 116 *et seq.*

Toxicity and Fatal Dose. The active principles upon which ergot depends for its action are not thoroughly understood. Ergot contains at least ten alkaloids, of which the most important, pharmacologically, are ergotoxine, ergotamine, and ergometrine (ergonovine). It contains, also, certain pigments which are peculiar to itself but are physiologically inert, and a number of amines which are also found elsewhere and some of which (*e.g.*, histamine), are physiologically active. Ergot cannot be considered a very powerful poison so far as life is concerned ; the danger of it lies in the circumstances in which it is given.

The medicinal dose of prepared ergot is 5 to 15 grains. It is occasionally employed in larger doses to excite uterine action, but usually ergotine or one of the preparations of the active constituents is given instead of the crude drug. The dose of the liquid extract is 10 to 20 minims, and of ergotoxine $\frac{1}{120}$ to $\frac{1}{60}$ grain.

The minimal fatal dose is quite unknown. It does not readily cause death in one large dose, but its fatal operation appears to be more strikingly developed by its long-continued use in small or medicinal doses. There is a suggestion that fever and sepsis increase sensitivity to ergot.

Symptoms. These must be divided into those of acute poisoning by the drug and those of the chronic effects either of eating bread, etc., made with flour containing some proportion of affected flour or of long continued administration of ergot preparations. In doses of from half a drachm to two drachms, ergot in powder has caused nausea, vomiting, dryness of the throat, thirst, pain in the abdomen, slight purging, pain in the head, stupor, and dilatation of the pupils. There may be tingling in the hands and feet, great muscular weakness and cramps. The blood pressure is raised. There may be suppression of urine, prostration and coma. Hæmorrhages may occur. The symptoms of chronic poisoning consist essentially in gangrene due to disease of the blood vessels distributed to the parts, this disease in turn being due to the continued action of the ergot on the muscular coat of the arteries.[1] There may be disturbances of sensation, itching and formication and occasionally complete paralysis of certain of the sensory nerves. Spasm and cramps occur in the muscles and sometimes choreiaform or epileptiform attacks.

Post-mortem Appearances. Nothing at all characteristic in the tissues, but particles of the powder may be seen.

Analysis. The powder of ergot has a faint fishy smell ; this is specially observed when it is rubbed with a solution of potash. This alkali dissolves it in part, and the solution acquires a dingy red colour. In the form of tincture, alcoholic or ethereal, the peculiar fishy odour of the extract when treated with potash is well marked. This is owing to the liberation of trimethylamine. It may, however, be concealed by other odours. Sometimes small particles of ergot, presenting a pink-red colour in the dark external coat, may be detected in the sediment by the microscope. When ergot has been taken in powder, fragments of

[1] For report of case see Yater & Cahill, *J. Amer. Med. Assoc.*, 1936, 106, 1625.

it may be found scattered over the lining-membrane of the stomach or bowels; these may be identified by the characters described. The ethereal tincture of ergot, evaporated to a syrup yields a yellowish-coloured oil, which, if any of the colouring matter of ergot is present, acquires a reddish colour when heated with a solution of potash. It also evolves a fishy odour of trimethylamine.

Sclererythrin Test. (Erythrin is an acid dye soluble in ether and giving a purple colour with alkalies.) The extraction is made by the Stas-Otto process with 80 per cent. alcohol. Concentrate. Take up the residue with weakly acidified water containing a little alcohol. Shake out with ether. The solution is red. Evaporate the ether. Dissolve residue with sodium bicarbonate. The solution is violet-coloured, and on spectroscopic examination one strong band in the yellow and two weak bands in the green will be seen.

Acidify the solution and extract free erythrin with ether. Two bands will be seen on spectroscopic examination, one in the green and one in the blue.

The examination must be made at once, as the colour is not permanent.

Tests for the Alkaloids. It has been stated that the mixed alkaloids of ergot may be extracted by chloroform from the final aqueous acid extract in the Stas-Otto process.

To detect the alkaloids qualitatively the following reactions may be applied :—

1. Dissolve a small portion of the alkaloids in about 1 cc. of concentrated sulphuric acid, adding a trace of ferric chloride; the solution assumes an orange-red colour changing to deep red, while the margin appears bluish or greenish-blue.

2. Dissolve a small portion in a few cubic centimetres of glacial acetic acid in a test tube, adding a trace of ferric chloride. Add this solution cautiously to another tube containing the same volume of concentrated sulphuric acid, taking care not to mix the liquids. A brilliant violet or intense blue colour is formed at the zone of contact.

3. Add an ethereal solution of the alkaloids to nitric acid diluted with an equal volume of water; in presence of ergot an intense blue colour (permanent for two days) develops at the junction of the two layers.

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Poisoning by Cocculus Indicus (Levant Nut); Picrotoxin

Source and Method of Occurrence. This is the fruit or berry of the *Anamirta Cocculus* (*Levant Nut*), imported from the East Indies. The berry contains from 1 to 2 per cent. of a non-nitrogenous poisonous principle (*Picrotoxin*). The shell or husk contains no picrotoxin, but a non-poisonous principle called menispermin. The seeds, in powder or decoction, give rise to toxic symptoms. About 2 grains of cocculus will cause toxic symptoms and about $\frac{1}{3}$ grain of picrotoxin. There are few well-authenticated instances of this substance having proved fatal to man. Several men suffered from this poison in 1829 near Liverpool; each had a glass of rum strongly impregnated with cocculus indicus. One died that evening; the rest recovered.[1]

The berries have been added to beer to increase its intoxicating properties, Carel[2] has reported a fatal case of poisoning by adding picrotoxin to whisky to facilitate robbery.

Cocculus indicus has been frequently used for the malicious destruction of fish and game. In one instance referred to by Taylor, there was reason to believe that 270 young pheasants had been poisoned by grain soaked in a decoction of this substance. *Barber's poisoned wheat* for the destruction of birds owes its poisonous properties to cocculus indicus (Horsley).

Picrotoxin is a most effective antidote to barbiturate poisoning; but its action is liable to get out of control which is a serious objection to its use. The same objection applies to its use as a convulsant in schizophrenia.

Symptoms. Shortly after ingestion of the drug a burning pain is felt in the gullet. Eventually the pain passes to the abdomen. Salivation, nausea, and vomiting follow. The respirations become deeper and quicker, the blood pressure rises and the pulse is slowed. The pupil contracts. There is dizziness, confusion and drowsiness, which are soon followed by unconsciousness. There may be uncontrollable twitching before consciousness is lost; but after a period of delay, convulsions take place, first tonic and then clonic in nature, probably due to medullary excitation. Death occurs from paralysis of the medullary centres. In milder degrees of poisoning the patient reels and stumbles about, and talks like a drunken man. The unconsciousness and clonic convulsions serve to distinguish the effects of this poison from those of strychnine. There is a strong disposition to sleep, and at the same time wakefulness. There is a heavy lethargic stupor, with a consciousness of passing events, accompanied by loss of voluntary power.

Treatment. This should be on general lines but a barbiturate is said to be the best antidote to picrotoxin.[3] Alkaloidal precipitants are not of value.

Picrotoxin is rapidly destroyed in the body and no time should be lost in submitting samples for analysis. Only a small percentage of the drug is excreted in the urine.

Analysis. Picrotoxin will be found in the acid chloroform extract in the Stas-Otto process. Evaporate, boil the residue with water and animal charcoal, filter, and add lead acetate as long as a precipitate

[1] Traill's "Outlines."
[2] Merck's *Arch.*, 1904, 6, 215.
[3] cf. Kohn, Platt, & Saltman. *S. Amer. Med. Assoc.*, 1938, *111*, 387.

forms, avoiding excess. Filter, and shake the filtrate with freshly prepared lead hydroxide, which forms an insoluble compound with picrotoxin. The precipitate is filtered off, suspended in water, and decomposed with sulphuretted hydrogen. Filter off the lead sulphide and extract the picrotoxin from the filtrate by means of chloroform (*Palm's method*).

The following tests should be applied :—

1. A minute quantity of the suspected substance is evaporated to dryness with concentrated nitric acid, and the residue moistened with concentrated sulphuric acid. On the addition of an excess of potassium hydroxide, the presence of 1-10,000th of a gram (1-650th of a grain) of picrotoxin will be shown by the appearance of a brick-red colour.

2. Picrotoxin dissolves in concentrated sulphuric acid with a golden yellow colour which changes to violet and then to brown upon the addition of a trace of potassium bichromate. This test may be applied directly to the lead compound obtained in the extraction process. The colour with sulphuric acid is discharged by a drop of nitric acid.

3. A small particle of picrotoxin treated with two drops of an alcoholic (absolute) solution of benzaldehyde (1 : 1) and then with one drop of concentrated sulphuric acid becomes red. If the liquid is agitated it shows a reddish-violet (Melzer).

4. A drop of sulphuric acid is added, and when the yellow colour is distinct, 1 drop of a 20 per cent. alcoholic solution of anisaldehyde is added. A dark blue-violet ring is formed and develops to a stable pure blue. Many glucosides and alkaloids give red or purple colours, but the blue is said to be characteristic of picrotoxin.

5. *Physiological tests.* The extract containing cocculus indicus is intensely bitter, and soon produces in an animal peculiar symptoms. One-twentieth of a grain of picrotoxin kills rabbits with peculiar opisthotonic convulsions. Fishes when immersed in water containing this substance make strange, sinuous movements, and fall powerless on the side.[1] Röber found that frogs are affected with tonic and clonic convulsions, and a peculiar inspiratory tetanus, which causes an inflated state of the abdomen. He thinks that this special condition is characteristic of the administration of picrotoxin to these animals.

The action of picrotoxin may also be tested on crustaceans which are highly sensitive to its effects.

Picrotoxin crystallises in slender hexahedral prisms having a silky lustre, in feathery needles, or long slender needles according to conditions —concentration and rate of cooling. It is soluble in 150 parts of cold water, but is more soluble in boiling water, and the solution has a very bitter taste. When heated in a tube, picrotoxin, like digitalin, evolves an acid vapour. Hydrochloric acid dissolves it without change of colour. It is soluble in alcohol, ether, chloroform, and in amyl alcohol. It is not precipitated by most of the usual alkaloid reagents. Though not a glucoside, it reduces Fehling's solution.

Langley has shown that picrotoxin may be separated from many of the poisonous alkaloids by taking advantage of its peculiar chemical properties. It does not combine with acids to form salts, but readily

[1] *B.M.J.*, 1875, March 27th, *et seq.*

with bases. Thus water containing a small quantity of potash will dissolve one-sixth or one-eighth part of its weight of picrotoxin. Such an alkaline solution will readily yield most of any dissolved alkaloids to ether when this solvent is shaken with the solution; but if the liquid is strongly acidified, most of the alkaloids remain combined with the acid, while the ether shaken with the liquid entirely removes the picrotoxin. Thus, in examining beer supposed to be adulterated with cocculus indicus, the liquid should be acidified with hydrochloric acid, and then shaken with twice its volume of ether. The ethereal solution thus obtained, when spontaneously evaporated, leaves the picrotoxin in crystals. Langley states that by this process he has detected so small a quantity as 1-750th of a grain of picrotoxin in a pint of ale. The stomach of a cat which had been poisoned was treated with alcohol, and the solution evaporated to dryness. Acidified water was poured on the residue, and the picrotoxin with some organic matter was dissolved. The acid liquid was shaken with ether, and crystals of picrotoxin were obtained by the evaporation of the ethereal solution.[1]

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Poisoning by Colchicum autumnale (Autumn Crocus, Meadow Saffron)

Source and Method of Occurrence. The corm from which the dry extract is prepared, and the seeds of the plant from which the liquid extract and the tincture are obtained, are both in official use and are both toxic. The plant grows in England, but is not common. Its flowers are showy, and it would be risky for children to eat them, but cases of this form of accidental poisoning are rare. Several cases have occurred, however, from the injudicious use of the medicinal preparations.

Many quack remedies for gout consist essentially of parts of the plant. Blair's Gout Pills consist of the ground corms of colchicum. A man, *æt.* 50, took twenty-eight of these pills in the course of four and a half days. He had nausea, griping pains in the abdomen, diarrhœa, hiccough, and vomiting, followed by stupor, convulsions, twitchings, and death in fourteen days. The pills, which were hard, were found by Beyte to yield three-fifths of a grain of colchicine.[2] The tincture, five to fifteen minims, the extract from the corm, a quarter to one grain, and the liquid extract prepared from the seeds two to five minims, are the official preparations and doses.

The official tincture contains 0·03 per cent. of colchicine, dose 5–15 minims; the liquid extract 0·3 per cent., dose 2–5 minims; and the dry extract 1 per cent., dose $\frac{1}{4}$–1 grain. The wine is no longer official.

[1] *Pharm. Jour.*, December, 1862, p. 277.
[2] *Lancet*, 1881, 1, p. 368.

Toxicity and Fatal Dose. The toxicity of the plant is due to the presence in it of two poisonous alkaloids—*Colchicine* ($C_{22}H_{25}NO_6$). and *Colchiceine* ($C_{21}H_{25}NO_6$). These substances appear to exert a similar pharmacological action, but the former is the more toxic of the two.

It is difficult to estimate the fatal dose, but it would seem that a grain would be likely to produce fatal effects. 15 cc. of tincture (U.S.P.), containing 6 mg. of colchicine have proved fatal, though larger doses have been survived.[1]

Colchicine must be considered a very poisonous drug, the official dose (U.S.P.) being $\frac{1}{120}$ grain.

Three and a half drachms of the vinum seems to be the smallest recorded single dose which has proved fatal; death ensued on the fourth day.

Symptoms. Symptoms occur only after an interval of three to six hours. There is then a burning sensation in the throat and acute intestinal pain. Vomiting and diarrhœa follow associated with severe cramps and tenesmus, followed by collapse, and death may occur from this irritant action.

A characteristic effect of the drug, however, is that after the latent period a motor and sensory paralysis supervenes, which may cause death from respiratory paralysis. The delayed action is observed irrespective of the way in which the drug is given. Even if given by intravenous injection the latent period still occurs. It would appear that colchicine requires to be altered in the body to another compound such as oxycolchicine before it has any specific effect on the central nervous system.

An interesting effect of colchicine is its action on the chromosomes of growing cells whereby their genetic characters may be altered.

The elimination of the poison is very slow, so that there is a possibility of causing toxic effects by repeated small doses.

Death may occur early or be postponed for several days. In the latter case there may be periods of remission with sudden aggravation of symptoms.

A woman, *æt.* 56, for whom wine of colchicum had been prescribed, took by mistake an ounce of the wine of the seeds, in divided doses, in twelve hours. She suffered from nausea, violent and profuse vomiting, slight purging, burning pain in the throat, great thirst, cold, clammy skin, feeble pulse, pain in the stomach, and pain in the forehead. Inflammation of the stomach supervened, and the retching, vomiting, thirst and pain continued for three days. She then recovered.[2] In other cases profuse purging has been the most prominent symptom, followed by death from exhaustion.[3] In one instance, in which two ounces of the wine were taken, the symptoms did not come on for an hour and a half; then copious vomiting of a yellow fluid occurred, with severe pain in the abdomen, tenderness, tenesmus, and thirst. The patient died in forty-eight hours without being convulsed or manifesting any sign of cerebral disturbance.[4]

Treatment. There is no specific antidote to colchicine, and treatment must be on general principles. The stomach and lower bowel should be

[1] Goodman and Gilman, *Pharmacological Basis of Therapeutics*, 1943, p. 240.
[2] Kennard, *Amer. Jour. Med. Sci.*, 1857, p. 69.
[3] *Pharm. Jour.*, July, 1861, p. 45.
[4] *Vide* also *Lancet*, I, 1903, p. 1254.

repeatedly washed out with a watery solution of tannic acid, and a suspension of animal charcoal left in the stomach. Opium may be necessary to control the pain and the spasm of the bowel, and papaverin has been recommended in preference to opium for this purpose. Intravenous saline with glucose or other infusion fluids designed to combat shock should be administered, and artificial respiration is indicated if the respiration fails.

Post-mortem Appearances. Signs of acute gastro-intestinal irritation are usually seen, but there is nothing characteristic.

Analysis. Colchicine is extracted in the Stas-Otto process from the final acid aqueous solution by chloroform. On evaporation of the solvent a yellowish residue is left. To isolate as pure colchicine as possible from the residue, the best method is that of Autenrieth. The yellowish residue is extracted by warm water and filtered. On cooling, the filtrate is shaken with petroleum ether to remove the fatty and resinous substances and then shaken again with chloroform, or the colchicine is precipitated with tannic acid solution after shaking with the petroleum ether. The collected and washed precipitate of the tannic acid is mixed with freshly precipitated and washed lead hydroxide. This mixture of precipitates is dried, ground to a powder and then extracted with chloroform. Evaporation of the solvent leaves nearly pure colchicine.

Tests. (1) Colchicine dissolves in nitric acid (specific gravity 1·4) with a dirty violet colour, changing, when stirred, to brownish red and finally to yellow. Addition of fragments of solid potash or soda till alkaline produces an orange-yellow or orange-red colour.

(2) Colchicine dissolves in strong sulphuric acid with an intense yellow colour. A drop of nitric acid added produces a green, blue, violet and then pale yellow colour. The addition of a fragment of caustic soda or potash brings out an orange-red colour.

(3) *Zeisel's Reaction.* Concentrated hydrochloric acid dissolves colchicine with an intense yellow colour. The colour is deepened by the addition of two drops of ferric chloride solution and boiling for two or three minutes. On cooling the solution and diluting with an equal volume of water, the liquid assumes a green or olive-green colour. If the liquid is shaken with a few cubic centimetres of chloroform, the solvent acquires a yellowish-brown or garnet-red colour. The aqueous layer retains its green colour.[1]

(4) One part of ammonium vanadate dissolved in two hundred parts of sulphuric acid produces a green coloration (sometimes very evanescent, and not distinct except with the pure alkaloid), which changes to a brownish violet; the reagent should be freshly prepared.

(5) An aqueous solution, allowed to stand for some hours with manganese dioxide and dilute sulphuric acid, and then filtered, becomes blue on addition of ammonium hydroxide in excess.

Ogier was able to obtain the reactions of colchicine isolated by the usual process from the exhumed bodies of dogs which he had poisoned with it five and half months before. In the bodies of animals poisoned with it, Oblonski detected colchicine four and a half months after death (Mann).

[1] Sydney Smith, "Forensic Medicine."

Poisoning by Conium maculatum, Coniine

Source and Method of Occurrence. The spotted hemlock is a very common plant indigenous in Great Britain. Its poisonous properties reside in the seeds, leaves, and roots. It belongs to the natural order Umbelliferæ.

The official "suicide" of Socrates was brought about by the use of hemlock, and it appears to have been the principal ingredient in the poison administered by the ancient Greeks to criminals.

In a case which was the subject of a trial for murder,[1] a child died in one hour after swallowing part of a teacupful of a decoction of hemlock, alleged to have been administered by the mother. The child supped the decoction until it lost the power of holding the cup; it became insensible and paralysed, and died in the chair in a sitting posture. There were no morbid appearances, and no hemlock leaves were found in the stomach, these having subsided in the cup. The child had been poisoned by the upper stratum of clear liquid. The mother was acquitted for want of proof of administration, the death of the child having taken place in secrecy.

Very few cases are recorded of poisoning by this plant, and most of them are of an accidental nature.

A medical electrician suffering from facial spasm took (beginning four hours after the last of a previous series of divided doses of a fluid extract of conium amounting in the aggregate to one hundred and eighty drops) at 4.10, 4.40, and 5.15 p.m., fifty minims (one hundred and fifty in all) of "Squibb's Fluid Extract." The first dose produced dizziness and muscular relaxation; the second, great muscular weakness, inability to stand, and thickening of speech, without relief of the spasm; the third, immediately, some nausea, and tremors about the chest. At 6.10 p.m. there were nausea, intense muscular weakness, partial dropping of the eyelid (ptosis, a common symptom), double vision, and great difficulty of speech. The pulse was sixty. Shortly after this he became unable either to speak or to swallow. He made signs for electricity, and, on being asked whether the direct or the faradic current, indicated the latter, and also the place of application of the electrodes, but was unable to hold one of the latter. Shortly after this he dropped back dead.[2]

David[3] records the following case:—

A family after eating a meal of perch with parsley sauce suffered from the following symptoms: headache, colic, diarrhœa, vomiting, fever, and delirium. Five persons in all were affected, and the symptoms commenced within an hour or two after the meal. Three adult patients had blood in their stools and rapidly became unconscious. The pupils were fully dilated, and showed no reaction to light. The temperature was raised and the pulse increased. One patient died on the following morning with symptoms of paralysis of the respiratory centre. The other two patients recovered, but suffered from continued diarrhœa and weakness of the limbs for some time. Two children who had eaten only a small quantity suffered slightly from diarrhœa and vomiting. The cause of the illness was proved to be *Conium maculatum* which was found growing in the midst of a bed of parsley, and portions of this plant were also found in the stools of the patients. These circumstances made the definite diagnosis easy, together with the fact that the fish eaten were known to have been quite fresh.

Toxicity and Fatal Dose. The poisonous properties of hemlock are due to coniine, which is associated in the plant with two nearly allied alkaloids, methyl coniine and conhydrine. All the alkaloids are toxic and have a similar curare-like action. Coniine, however, is quantitatively the most important, and is also of interest as being the first alkaloid to be synthesised (Ladenburg, 1886). Its action is almost entirely on the

[1] *R. v. Bowyer*, Ipswich Sum. Ass., 1848.
[2] *The Sanitarian*, June, 1875.
[3] *Zeitschr. f. Medizinalbeamte*, No. 20, 1913.

nervous system, and death is brought about by this action being exerted on the vagi or nerves of respiration, or by paralysis of the centres in the medulla.

Duration. The symptoms come on after a variable interval required for digestion and absorption, and rather rapidly, *i.e.*, in three or four hours, increase in severity, and kill by involvement of the cardio-respiratory nerves.

Symptoms. As in poisoning by most plants, if portions be eaten there may be symptoms (vomiting, nausea, diarrhœa) of gastro-intestinal irritation before the more specific effects of the coniine are exhibited.

The characteristic effect produced by hemlock is a progressive muscular paralysis. A peculiar muscular debility sets in ; the gait is staggering, the lower limbs become weak and eventually paralysed ; the paralysis advances upwards, eventually reaching the respiratory muscles. There is dyspnœa, anxiety in the region of the heart, and occasionally convulsions. The pupils are dilated, though not to the same extent as when a solanaceous plant has been taken. When the respiration is affected there is marked blueness of the surface of the body. Death takes place from respiratory paralysis.

A man ate a large quantity of hemlock plant by mistake for parsley. In from fifteen to twenty minutes there was loss of power in the lower extremities ; but he apparently suffered no pain. In walking, he staggered as if he were drunk ; at length his limbs refused to support him, and he fell. On being raised, his legs dragged after him, and when his arms were lifted they fell like inert masses, and remained immovable. There was complete paralysis of the upper and lower extremities within two hours after he had taken the poison. There was a loss of the power of swallowing, and a partial paralysis of sensation, no convulsions, but only slight occasional motions of the left leg ; the pupils were fixed. Three hours after eating the hemlock the respiratory movements had ceased. Death took place in three hours and a quarter ; it was evidently caused by gradual asphyxia from paralysis of the muscles of respiration ; but the intellect was perfectly clear until shortly before death.

Treatment. Empty the stomach, wash out with a solution of permanganate of potash and leave a suspension of animal charcoal in the viscus. Give general stimulants, alcohol, coffee, etc. Of antidotes, strychnine is the best, but the antagonism is not absolute ; it should be given subcutaneously in doses of $\frac{1}{30}$ grain, repeated if necessary in an hour. Artificial respiration and the administration of oxygen and carbon dioxide may be of value.

Post-mortem Appearances. Beyond the possibility of fragments of the plant being found in the stomach, there are few, if any, *post-mortem* indications of any value. In a case described above, the stomach was found to contain a green-coloured pulpy mass resembling parsley. The mucous coat was much congested, especially at its greater end, where there were numerous extravasations of dark blood below the membrane, over a space of about the size of the hand. The intestines were healthy, here and there presenting patches of congestion in the mucous coat. The blood, throughout the body, was fluid, and of a dark colour. A portion of the green vegetable pulp was identified as part of the leaves of the *Conium maculatum*. Some of the leaves bruised in a mortar, with a solution of potash, gave out the peculiar odour of the alkaloid coniine,[1] which is reminiscent of that of mouse urine.

[1] *Edin. Med. and Surg. Jour.*, July, 1845, p. 169.

Analysis. Hemlock is known from most other plants which resemble it by its large round smooth stem, with reddish spots. The leaves are of a dark-green colour, smooth and shining. Every portion of the plant has a peculiar and disagreeable smell when rubbed or bruised, resembling that of cats' urine, or, according to some, the odour of mice. It is strongly brought out when the stem, leaves, or seeds are rubbed with a solution of caustic potash. The *seeds* of hemlock are peculiar in their form, and are easily distinguished from the seeds of other umbelliferous plants.

The determination of the presence of fragments of leaves in poisoned liquids, and in the contents of a stomach, may be of importance in evidence. The appearance and smell of the leaves, either when bruised or when rubbed with a solution of potash, will greatly aid a medical witness in forming a judgment, as there are many umbelliferæ which bear a close resemblance to hemlock in the form of their leaves.

Coniine resembles nicotine and ammonia in its liquidity, alkalinity, volatility, and in some of its chemical properties. It is a liquid of oily consistency, usually of a pale yellow colour, but is colourless when freshly prepared, powerfully alkaline, and has, when its vapour is diluted, a smell resembling that of mice, and an acrid, bitter taste. It gives a volatile greasy stain to paper, and burns with a yellow flame and thick smoke. 1. It is scarcely coloured or affected by nitric, sulphuric, or hydrochloric acid ; the last-mentioned acid produces with it dense white fumes of hydrochloride of coniine, and on heating the mixture this salt remains in prismatic crystals. 2. It dissolves in one hundred parts of water. 3. It is soluble in alcohol and ether, and this last-mentioned liquid removes it from its aqueous solution, and leaves it in oily globules on evaporation. 4. It gives a white precipitate with corrosive sublimate. 5. It gives a yellow precipitate with phospho-molybdic acid. 6. A solution of iodine in potassium iodide gives a reddish-brown precipitate. 7. It gives crystalline precipitates with auric chloride and platinic chloride in concentrated solutions only. 8. Tannic acid gives a dingy white precipitate. 9. Gallic acid gives no precipitate, but slowly acquires a yellowish colour. 10. If coniine is warmed with potassium bichromate and dilute sulphuric acid, butyric acid is produced, which may be recognised by its characteristic odour (Luff). 11. If coniine is dropped into a solution of alloxan, the latter is coloured an intense reddish-purple after a few minutes. On standing, white needle-shaped crystals separate out. These, if collected and dissolved in cold caustic potash solution, produce a bluish-purple colour, and emit a strong odour of the coniine (Luff). 12. Warmed with a little chloranil in benzene solution, coniine gives a green colour and on evaporation crystals separate, green at first but rapidly turning blue on exposure to air. Its odour and insolubility in water, as well as several of the characters above mentioned, serve to distinguish coniine from nicotine and ammonia, but it may be readily separated from ammonia by potassio-mercuric iodide, which precipitates it even more completely than tannic acid. In reference to its presence in *organic mixtures*, it may be detected by its peculiar odour, or by distilling the liquid with a solution of potash, and examining the distillate extracted from organic mixture. It may be extracted by the method detailed on p. 265 *et seq.*

The reactions produced by tests on small quantities are not very conclusive and must be confirmed by evidence of the action of the poison

on the body from the symptoms. As in reference to strychnine, veratrine, and other alkaloids, an incautious analyst may readily come to the conclusion that he has found " traces," and ascribe death to the poison.

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Poisoning by Croton tiglium (Croton Oil)

Source and Method of Occurrence. Croton oil is extracted from the seeds of the *Croton tiglium*. The seeds, which are sometimes called *Purging nuts*, resemble castor seeds in size and shape. They have a dark brown or blackish colour, and are marked by longitudinal lines, and have no smell. Their taste is at first mild and oleaginous, afterwards acrid and burning. When heated they evolve an acrid vapour. The seeds contain in addition to the oil and croton resin, which is the active component of the oil, a toxalbumin similar to ricin which is not expressed with the oil but which is highly toxic.

Toxicity and Fatal Dose. Croton oil is a powerful drastic purgative, producing, in a large dose, severe purging, collapse, and death. Its dose is given as from half to one or two minims, but the preparation is no longer official.

In man, a dose of from fifteen to twenty drops of the *pure* oil might give rise to excessive purging, and cause death by exhaustion. The cases recorded of its fatal operation are few, and do not enable us to solve this question from observed facts. According to Landsberg,[1] thirty drops of the oil have killed a dog, and Christison states that he has known four grains of the oil to produce an alarming degree of purging. It is frequently mixed with castor oil and other substances, and the presence of these must, of course, influence the dose required to act fatally.

The smallest recorded fatal dose seems to be the following : —A child thirteen months of age, died in six hours from a small dose given b mistake. The croton oil was mixed with soap liniment, and the quantity taken was supposed to be **less than three minims** of the oil.[2]

Duration. Croton oil is occasionally used medicinally when a rapid and powerful effect is required, and symptoms commonly set in within half an hour. It is now very rarely used, as its drastic action renders it dangerous. In large doses it acts immediately and death may occur in four or five hours.

Symptoms. These are directly exhibited by the alimentary canal, as might be expected from the medicinal use. In a case where a man swallowed by mistake two drachms and a half of croton oil, in three-quarters of an hour the surface was cold and clammy, the pulse imperceptible, the breathing difficult, and the extremities and face were as blue

[1] Christison's " Dispensatory."
[2] *Med. Times and Gaz.*, 1870, 2, p. 466.

as in the collapsed stage of cholera. Within an hour and a half purging set in; the stools were passed involuntarily, and the abdomen was very sensitive to the touch. The patient complained of a burning pain in the course of the gullet. He died in four hours after swallowing the poison. There was no marked change in the mucous membrane of the stomach. In another case a druggist swallowed, by mistake for cod-liver oil, half an ounce of croton oil. He felt a burning sensation in the throat and stomach, soon followed by vomiting and copious purging, with symptoms of collapse. He did not recover until after a fortnight. A girl, *æt.* 19, took by mistake a teaspoonful of a liniment consisting of equal parts of croton and olive oils. In about half an hour she complained of an intense burning sensation in the throat and gullet but no pain in the stomach. Her pulse was 84. Vomiting came on in a severe form, and this was promoted by a zinc emetic and warm water. After the vomiting had continued for a quarter of an hour, she complained of a severe pain in the stomach. Purging was not a prominent symptom. A day or two afterwards she recovered.[1] In another case, a girl, six years old, took by mistake about fifty-five drops of croton oil. There was vomiting, with some purging and feverishness for three or four days, but the patient recovered.[2] In these cases it is not improbable that the oil may have been adulterated.

Treatment. Must be carried out on general principles (*vide* pp. 318 *et. seq.*).

Post-mortem Appearances. Signs of irritation may be met with in the alimentary canal, but nothing characteristic of croton oil.

Analysis. Discharges, etc., are acidified with tartaric acid and extracted with ether. The ether is allowed to evaporate. The residue, if rubbed on the inner side of the arm, produces a reddened area which may blister. It may also be administered to animals and the gastro-intestinal symptoms noted. There are no reliable chemical tests for small quantities.

Cases. A case was tried at Liverpool[3] in which the accused were charged with having caused the death of a man by placing in food, of which he and others had partaken, two drachms of powdered jalap, and from two to six drops of croton oil. Several persons, including the deceased, suffered from vomiting and purging; but they recovered, and the deceased himself so far recovered as to be able to go about as usual. He was subsequently attacked with inflammation and ulceration of the bowels, from which he died. The accused were acquitted, as the medical evidence at the trial failed to make out the connection of this subsequent illness with the jalap oil which had been put into the food.

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[1] *Edin. Med. Jour.*, August, 1861, p. 134.
[2] *Lancet*, 1870, 1, p. 553.
[3] *R.* v. *Massey and Ferrand.*

Poisoning by Curare

Source and Method of Occurrence. Curare is the active principle obtained from species of strychnos, cocculus, and other plants growing in South America and elsewhere. In the places where these plants grow it has long been known as one form of arrow poison (*vide* p. 646), killing with only a small wound. In England it was until recently almost confined to laboratory experimental work, though a hypodermic injection of it had been suggested for tetanus, hydrophobia, strychnine poisoning, etc. Recently it has been used in anæsthesia to obtain complete muscular relaxation and in shock therapy to prevent violent muscular contractions.

Cases of poisoning by it are very rare ; there is one recorded case of its use for suicidal purposes. In 1917 there was a frustrated attempt on the life of the Prime Minister of England by means of this drug.[1]

Toxicity and Fatal Dose. It is said to be practically innocuous when swallowed (like snake venom), requiring to be injected into the blood without going through the stomach.

Its chief effect is to paralyse all voluntary movement by its action on the connection between the puerperal nerve end and the striated muscle fibre. It is probable that this action is due to interference with the production of acetylcholine or with its action on the muscle fibre. Curare acts first on the muscles of the toes, then on the limbs, head and neck, and lastly on the muscles of respiration, causing death by asphyxia.

Symptoms. Increasing paralysis with the ordinary effects on circulation and respiration are the symptoms to be expected.

Treatment. Its effect on the myoneural junctions may be antagonised by the use of physostigmine or better by neostigmine in doses of 2 cc. of a 1 in 2,000 solution intravenously. Artificial respiration may be required for long periods.

Analysis. In most chemical tests it resembles strychnine, but sulphuric acid alone imparts a violet colour to solutions of curare, while it has no effect on strychnine. The active principle of curare is probably an alkaloid, curarine. Curine, tubocurarine, protocurine and protocurarine have also been described. With strong nitric acid curarine gives a deep red or purple colour. With concentrated sulphuric acid it gives a lasting deep blue or violet colour. Curarine can be distinguished from strychnine by the colour that it gives with nitric acid or with sulphuric acid, and also by the fact that potassium bichromate produces an amorphous precipitate of curarine chromate, whereas strychnine chromate is a crystalline precipitate (Luff).

It should be noted, however, that of these various alkaloids, only curarine gives the blue or violet colour with concentrated sulphuric acid. Mitchell (Allen's Commercial Organic Analysis," 5th Ed., vol. VII., p. 773), states that the modern commercial "Curarine" is the hydrochloride of tubocurarine, whereas gourd curare, from Strychnos toxifera (of which curarine is the main alkaloid), has practically disappeared from commerce. Tubocurarine has an intensely bitter taste, gives no colour with sulphuric acid alone, but a blackish colour on further addition of potassium bichromate, and gives a voluminous yellow precipitate with

[1] Birkenhead, "Famous Trials of History," 1926, p. 219.

metaphosphoric acid. It is less poisonous than curarine. The free base is a reddish-brown syrupy liquid (easily decomposed), the hydrochloride an amorphous yellow-red powder.

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Poisoning by Cytisus laburnum

Source and Method of Occurrence. The common laburnum, a species of cytisus, though not indigenous in England, is so frequently grown in gardens and shrubberies, and has such showy flowers and fruit, that it not infrequently gives rise to cases of poisoning in children, who accidentally eat the seeds or flowers.

Toxicity and Fatal Dose. The toxicity of the laburnum tree (chiefly bark and seeds) depends upon the presence of an alkaloid, cytisine, which seems to be the same as ulexine, the alkaloid present in the common furze (the prickly nature of this plant prevents accidents). The symptoms are closely similar to those of nicotine.

Three or four of the seeds seem to be capable of giving rise to very alarming symptoms, but the minimal fatal dose is undetermined.

Cytisine is said to be the poison contained in an insect powder which is known by the name of Australian or Persian insect powder.

Duration. In the reported cases the symptoms appeared after varying intervals of an hour or more, and passed off slowly.

Symptoms. Pain in the stomach, nausea, vomiting and depression, are followed by mental confusion, giddiness, muscular weakness and inco-ordination. Convulsions clonic in character, indicating an action on the medulla may occur. This stimulation is short-lived, and is followed by depression and paralysis of the medullary centres.

A girl, *æt.* 18, idly and unthinkingly, put a small portion of a laburnum branch into her mouth, carrying it for some hours, and chewing it. It was described as of the thickness of the little finger, and two or three inches long. There were some yellow flowers with it, but she was not aware that she had swallowed any. In about half an hour she felt unwell, but she was not seen by a medical man until the day following. The symptoms then were great pain in the stomach, nausea and retching, but no vomiting; pulse 100, tongue white, great thirst, anxiety and pallor of countenance, dilated pupils, sense of fainting even while lying down, and great exhaustion.

There was no purging. Under treatment these symptoms disappeared, and the girl recovered within about a fortnight.[1]

Two boys swallowed a quantity of laburnum seeds in a cake. Within about three-quarters of an hour one was seized with vomiting and purging, weak and frequent pulse, severe rigors, muscular twitchings in the face and neck, and great epigastric pain. The pupils were dilated, but there was no headache. Many seeds were vomited. There was a great disposition to sleep, and coldness of the skin. Under treatment they recovered.[2] A girl, *æt.* 9, died in consequence of having eaten a

[1] *Lancet*, 1870, 2, p. 182.
[2] *Pharm. Jour.*, October, 1862, p. 185.

few of the seeds. A boy, *æt.* 4, ate about ten of the seeds. In half an hour he began to vomit, the vomited matter consisting of food and thick mucus. He afterwards became drowsy, and was seized with convulsions, shaking violently, and drawing up his limbs at intervals. Although drowsy, he was easily roused, but soon dozed off again. Both pupils were widely dilated, pulse eighty-five and small, surface, especially of the limbs, cold. He fell into a calm sleep, and the next day he was well.[1]

A case of poisoning by the *bark* was the subject of a trial at Inverness.[2] A youth with the intention of merely producing vomiting in one of his fellow-servants, a young woman, put some dry laburnum bark into the broth which was being prepared for their dinner. The cook, who remarked a "strong peculiar taste" in the broth, soon became very ill, and in five minutes was attacked with violent vomiting. The account of the symptoms is imperfect, for the cause of them was not even suspected until six months afterwards. The vomiting continued thirty-six hours, was accompanied by shivering, pain in the abdomen, especially in the stomach, great feebleness, and severe purging. These symptoms continued, more or less, for a period of eight months; and the woman fell off in flesh and strength. At this time she was seen by a physician, who had been called on to investigate the case. She was then suffering from gastro-intestinal irritation, vomiting after food, pain in the abdomen increased by pressure, purging, tenesmus and bloody evacuations, with other serious symptoms. The medical opinion was that she was then in a highly dangerous state. The woman eventually recovered. There was no doubt, from the investigation made by Ross and Christison, that her protracted illness was really due to the noxious effects of laburnum bark.

Treatment. Emetics and the stomach-tube must be used to empty the stomach, and the depression and collapse treated on general principles. The bowel must be washed out to get rid of parts of the plant or seeds. Artificial respiration must be persisted in, for death is caused by asphyxia.

Post-mortem Appearances. Nothing characteristic unless portions of the plant are obtainable to examine with the eye and lens.

Analysis. The urine and vomitus are of especial importance for analysis, as the alkaloid is rapidly excreted by the kidneys. The modified Stas operation described on p. 265 must be utilised for the extraction of alkaloids. Cytisine is a strong base, and it is therefore necessary to make the liquid very definitely alkaline with sodium hydroxide before attempting to extract the alkaloid by means of organic solvents. Radziwillowicz recommends amyl alcohol for this purpose, but Moer and Plugge state that the pure alkaloid is much more soluble in chloroform than in amyl alcohol. It is insoluble in ether.

With a solution of a ferric salt cytisine yields a red colour, which disappears on the addition of a few drops of a solution of peroxide of hydrogen; on subsequent warming a blue colour is produced. This test is very delicate; according to Moer and Plugge it will indicate the presence of 0·05 mgrm. of the alkaloid.

Nitrobenzene containing dinitrothiophene produces a red-violet coloration with cytisine.

If thymol is added to cytisine dissolved in concentrated sulphuric acid and the mixture is heated, a yellow colour appears and develops into an intense red.

Cytisine dissolves in concentrated sulphuric acid without undergoing change of colour; on warming, the mixture becomes yellow. If to a little cytisine dissolved in a few drops of concentrated sulphuric acid in

[1] *Lancet*, 1871, 2, p. 396.
[2] *Edin Med. and Surg. Jour.*, October, 1843, p. 300.

the cold a drop of nitric acid is added, a yellow colour is produced. If to a mixture of cytisine and sulphuric acid a fragment of potassium dichromate is added, a yellow colour is produced which changes to dirty-brown and finally to green.

If cytisine dissolved in a drop of fuming nitric acid, is evaporated to dryness and the orange residue is moistened with alcoholic potash (Vitali's test), a green colour is produced at once and changes to a dull violet.

The *seeds* are somewhat kidney-shaped, and slightly hooked at the hilum. They shrink in drying, become dark-coloured, and present irregular depressions on the surface. They have no markings, and are thus easily distinguished from most other poisonous seeds. They are larger than those of *Datura stramonium*.

Cases. The following six cases of poisoning by *Cytisus laburnum* are recorded by St. Johnston. W. G., aged ten years, was brought to the hospital on August 20th, 1891, and stated that he, together with several other boys, that afternoon, about two hours after dinner, had found during a walk a tree with pods growing on it, which they thought to be small beans. Several of these pods were found to be the seed-cases of the *Cytisus laburnum*. W. G. had eaten the contents of four pods (that is, eight or ten seeds). Five or ten minutes afterwards he began to sweat, but soon became cold and shivering, and a little later was seized with vomiting. He vomited three times, and there were portions of the seeds in the matter ejected. He was then purged once. About an hour later he came to the hospital, and he was cold and shivering, skin pale, and pulse scarcely perceptible —in fact, in a state of general collapse. His pupils were dilated, and he felt very drowsy and giddy, but suffered no pain. After his stomach had been well cleared by emetics, brandy was administered. He, however, showed little signs of improvement for the next hour or so, when an enema of hot strong coffee was given, and the patient wrapped in blankets, and hot bottles applied. The collapse then gradually passed off, and the patient was able to leave the hospital in a few hours. He has had no bad symptoms since. P. H., aged eleven years, was brought in a short time after the previous case, and in a similar condition. He had eaten the seeds of three or four pods, and some time after was seized with vomiting, great collapse, shivering, giddiness, and scarcely perceptible pulse; his pupils were widely dilated. This child appeared to be in a critical condition, and was admitted as an in-patient, put to bed, and treated as in the previous case. S. H., aged seven years, a much smaller boy, had eaten the seeds of several pods. In this case also the collapse, shivering, giddiness, vomiting, and extreme weakness of pulse, and dilated pupils, were so marked as to necessitate his admission. After similar treatment he rallied, and felt quite well next day. In these three cases there was marked collapse with very small weak pulse, shivering, giddiness, vomiting, and widely dilated pupils, but no pain. There was also marked drowsiness in all three. In the two cases admitted the temperature remained subnormal for two days. A. R., aged nine years, was brought up some hours later, suffering from purging and vomiting. There was little or no collapse. He had been with the other children, and had eaten the contents of one (?) pod only. He required little treatment, and was not detained. In this case purging was the chief symptom. A. B., a little girl, five years old, had taken one seed only. She was sick, but showed no other symptoms. E. G., a girl, aged seven years, had eaten one seed only, which her brother had given her, but as she showed no symptoms two hours afterwards she required no treatment.[1]

The *flowers* of this plant are highly noxious. A child, between three and four years of age, ate twelve laburnum flowers, and in about fifteen minutes it complained of sickness and severe pain in the stomach. The child vomited a quantity of mucus mixed with the yellow petals of the laburnum. An emetic was given; this cleared the stomach, and the child recovered. There was no purging.[2]

[1] *B.M.J.*, 1891, 2, p. 695.
[2] Guy's Hosp. Rep., 1850, p. 219.

For cases in which five children were poisoned by laburnum seeds, *vide B.M.J.*, 2, 1895, p. 778, and three non-fatal, but very serious, cases are reported in the *Lancet*, 2, 1901, p. 491.

In 1882, two fatal cases of poisoning occurred by some undetermined portions of the laburnum tree.[1] The victims were two children, aged respectively three and eight years. The elder child was seized with vomiting and diarrhœa, headache, and prostration. In six hours the vomiting and diarrhœa ceased. She then made noises in her breathing, and continued in much the same state till her death, fourteen hours after she was attacked. Next day the younger child became tired and sleepy, vomited, and complained of headache. She vomited freely, and passed two motions. Five and a half hours after the commencement of the symptoms she was convulsed, and the convulsions continued till her death, eight hours from the commencement of the attack. On *post-mortem* examinations being made, some signs of irritation of the stomach and intestines were observed in each case.

The contents of the stomachs of both children were examined by the eye and by the microscope, but no fragments of the structures of the laburnum were discovered. Evidence was, however, obtained by Fairley, in both cases, of the presence of cytisine, the poisonous alkaloid present in the flowers, seeds, bark, leaves, and all parts of the common laburnum tree. A small portion of an alcoholic extract made from the stomach and contents of the elder child was administered to a mouse, which died in the course of a few hours.

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Poisoning by Daphne mezereum

Source and Method of Occurrence. *Daphne mezereum*, or *mezereon*, occasionally gives rise to accidental poisoning in children who pluck and eat the berries. It is very rare in England and doubtfully indigenous, but is found planted in shrubberies. The juice is strongly irritant, and tends to destroy mucous surfaces with which it comes in contact.

The symptoms are illustrated by the following cases: Eagar saw a child, four years old, after it had eaten at least twelve mezereon berries. Convulsions occurred before any other symptoms; an emetic was given, and vomiting procured; three hours after, the lips and tongue were swollen; the tongue, twice its natural size, was raw, and protruded beyond the lips; there was difficulty in swallowing, the limbs were cold, and the pulse—one hundred and thirty in the minute—was very weak; recovery took place. Dunn saw a child of the same age which had also eaten some mezereon berries. It was restless, and complained of pain in the mouth and throat; vomiting took place spontaneously before the child was seen; an emetic was afterwards given which brought away further portions of the berries. The child was drowsy, prostrate, pale in the face, with dilated pupils, scarcely perceptible pulse, and cold limbs; the mucous membrane of the tongue and of the roof of the mouth was white from the action of the acrid juice of the berries; the child recovered.

Treatment. Evacuate the stomach, and afterwards administer an aperient, with such further treatment as the symptoms require.

[1] *B.M.J.*, 1882, 1, p. 199.

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Poisoning by Digitalis purpurea

Source and Method of Occurrence. The purple foxglove, *Digitalis purpurea*, is a well-known common wild flower in England. It belongs to the natural order Scrophulariaceæ. All parts of the plant are poisonous, but only the leaves are used for medicinal purposes.

Owing to its value as a medicine digitalis has been more closely studied than any other of our English plants. The result has been to isolate several glycosides, including digitoxin, gitalin and gitoxin. Many chemists have worked on the active principles of digitalis, each giving a name to his own discovery, but the resulting confusion has been greatly clarified in recent years.[1] Digitoxin may be looked upon as the representative member of the group. In general it is inadvisable to use the term digitalin, for this indicates not a pure substance but a mixture of variable constitution. Nativelle's " digitalin " consists almost entirely of digitoxin.

The leaves may be given in doses of one-half to one and a half grains for repeated medication or three to ten grains for a single dose; the infusion, ninety to three hundred minims repeated or one to four ounces single; and the tincture, five to fifteen minims for repeated and thirty to ninety minims for a single dose.

Digitalis poisoning is not common, but overdoses given medicinally but injudiciously, have produced unpleasant symptoms, and possibly hastened the end in many cases of heart disease. Sir Thomas Stevenson saw a patient under the influence of digitalis die very suddenly on being raised by the nurse from the recumbent to the sitting posture.

Probably many cases occur from using different active principles with the same name but different composition.

In 1864, a man was executed in Paris for murder by digitalin.

Toxicity and Fatal Dose. Thirty-eight grains of the leaves and nine drachms of the tincture have proved fatal, so that the plant and its official preparations cannot be considered as very fatal poisons, but digitalin operates powerfully on man and animals and must be regarded as an active poison. Homolle found in experiments on himself that small doses of digitalin taken at intervals lowered the pulse to about one-fourth or one-fifth of the normal standard: thus in himself it fell to seventeen in one minute, which represents a fourth of the normal pulsations. In doses of from one-fifteenth to one-thirtieth of a grain in twenty-four hours, digitalin slackened the circulation. In doses above one-fifteenth of a grain, it produced on adults emetic and purgative effects, sometimes suddenly, at others slowly and gradually. In doses of from one to two grains, unless speedily removed by vomiting, it killed dogs in a few hours.[2]

[1] Elderfield, " Chemistry of the Cardiac Glycosides," *Chem. Rev.*, 1935, *17*, 187. Jacobs, *Physiol. Rev.*, 1933, *13*, 222. Stoll, " The Cardiac Glycosides," Pharm. Press, London, 1937.

[2] Orfila, " Toxicologie," vol. 2, p. 350; see also a paper by Fagge and Stevenson, Guy's Hosp. Rep., 1866, p. 37.

It is found that digitalis is more rapidly absorbed from the stomach than excreted by the kidneys, and hence it has been called a **cumulative** poison. Nearly half the amount of digitoxin injected in a single dose is still present after five days.[1] Sometimes symptoms of poisoning occur very suddenly in patients being treated with the drug on account of this cumulative action.

Symptoms. The effects from a pharmacological point of view have been very carefully studied, and it has been found to have three principal actions: (1) it is an irritant to the stomach; (2) it slows and makes more forcible the heart-beat, improves the circulation in the heart muscle, and has a beneficial effect in auricular fibrillation; (3) it increases the tone of the arterioles and so increases blood pressure.

If toxic symptoms occur by overdose or cumulative action, they are first indicated by depression, nausea, vomiting, headache and giddiness, with some cardiac discomfort. The toxic effect on the heart is shown by an excessive slowing of the heart and increased strength of contraction. The rate may drop to twenty or so beats per minute. There may be extra-systoles which cannot be felt at the wrist, but which may be heard by the stethoscope. In the final stage of poisoning the heart becomes rapid and irregular, there are fibrillary twitchings, and it stops in systole. Death usually occurs quite suddenly.

The drug may exert a stimulating effect on other organs, such as the gastro-intestinal tract, producing diarrhœa, and stimulation of the uterus leading to abortion.

After very large doses, convulsions similar to those caused by picrotoxin may be seen.

A young man swallowed a strong *decoction* of foxglove by mistake for purgative medicine. He was soon seized with vomiting, pain in the abdomen, and purging. In the afternoon he fell asleep. At midnight he awoke; was attacked with violent vomiting, colic, and convulsions; the pupils were dilated and insensible to light; his pulse was slow and irregular. He died twenty-two hours after taking the poison.[2] A few grains of the powdered leaves have been known to produce giddiness, languor, dimness of sight and other nervous symptoms. A drachm, however, has been taken without causing death; but in this instance it produced the most violent vomiting. A woman made an infusion of digitalis, and swallowed it by mistake. The symptoms which followed were vomiting, paleness of the face, coldness of the skin, prostration, muscular feebleness, a persistent feeling of drunkenness, headache, giddiness, confusion of sight, dilatation of the pupils, and loss of sensibility. The vomiting was constant, and aggravated by anything that was taken. There was constipation of the bowels, with suppression of urine. There was thirst, with pains in the abdomen increased by pressure, and great restlessness at night. At first the pulse was fifty-two. On the fourth day it was forty-one to forty-two. On the fifth day it was fifty-eight, less irregular, and the symptoms had abated. During the night she got up, and on returning to her bed suddenly fainted, and died. A

[1] Dixon, "Manual of Pharmacology," 1925, p. 186; see also *Arch. Int. Med.*, 1937, *60*, 240.

[2] Wibmer, *op. cit.*, "Digitalis."

person labouring under symptoms of poisoning by digitalis should always be kept in the recumbent posture.[1]

Treatment. The stomach should be emptied and washed out with a solution of tannic acid. If parts of the plant have been taken, the bowel should be washed out. In clinical cases the main thing is the cessation of administration. In the stage of excessive slowness from vagal inhibition, atropine is of value. The patient must be kept at absolute rest on his back.

Post-mortem Appearances. Portions of the plant may be found in the stomach or bowel, and slight inflammation of the stomach owing to the irritant action of the drug may be observed.

In prolonged cases there may be focal necrosis or other pathological changes in the pupillary muscles and the endocardial surface of the left ventricle.

Extraction. The acid aqueous extract obtained in the usual way (p. 265) from organic matter is best shaken out with chloroform, in which all the active principles of digitalis are soluble ; they are not all soluble in ether nor in benzene ; it is to be remembered that digitalin in *acid* solution is taken up by chloroform.

Analysis. Fragments of leaves or seeds found in the stomach may be identified by the aid of the microscope. The *seeds* are reddish brown, remarkably small, oblong, and somewhat angular in shape and have peculiar markings. These characteristics make it easy to distinguish them from the seeds of other poisonous plants—hyoscyamus, datura, belladonna, etc.

In examination of the leaves or preparations from them as of extracts prepared from vomitus, tissues, etc., the physiological tests are of great importance. A full discussion of these will be found in *Jour. Pharm. Exp. Ther.*, 1926, 29, 407 (Lenz).

The methods recommended in the British Pharmacopœia (1932), p. 619, for the biological assay of digitalis are suitable for quantitative estimation in toxicological work, provided the identity of the poison has been ascertained. These methods consist of (1) determining the lethal dose in frogs, comparison being made with an extract from the standard preparation of digitalis ; (2) determining the volume of the extract which, injected at a slow uniform rate into the vein of a cat or guinea pig (under ether anæsthesia, and with artificial respiration), is just sufficient to arrest the heart ; this volume is then compared with the volume of standard extract necessary to produce the same effect.

(In both tests, of course, a number of animals must be employed.)

For the physiological *detection* of digitalis the effect of the drug on the ventricular rate and on the ventricular and auricular rhythm may be studied.

The chemical detection of " digitalis " may be somewhat complicated by the fact that besides the " official " preparations (powdered Digitalis and Tincture of Digitalis) many more or less purified glycosidal fractions from digitalis, strophanthus and squill are commercially available and

[1] *Edin. Month. Jour.*, 1864, p. 169.

these contain different glycosides as their chief constituent. These glycosides, though similar in physiological properties, differ slightly in their chemical reactions. Thus it is stated that Merck's digitalinum germanicum contains digitonin,[1] gitonin and digitalin ; Homolle's amorphous digitalin consists of digitalin with some digitoxin ; and Nativelle's crystallised digitalin is mainly digitoxin.

The digitalis glycosides are very sparingly soluble in water, but dissolve easily in hot alcohol. Digitoxin is readily soluble in ether and chloroform, but digitalin and digitonin are much less so. (It follows that in a chloroform extract from the mixed glucosides, as in the Stas-Otto process, the reactions of digitoxin may be expected to predominate.)

The glycosides are precipitated by tannic acid, but not by potassium mercuric iodide (distinction from alkaloids) ; when heated, they melt, decompose, and evolve an *acid* vapour, not an ammoniacal one ; they dissolve in concentrated nitric acid, and the solution rapidly becomes pale yellow ; they do not affect iodic acid.

Hydrochloric acid dissolves the glycosides. On heating, the solution of digitoxin becomes greenish brown ; that of digitonin (which is yellow in the cold) becomes a reddish violet with a faint green fluorescence.

Sulphuric acid (concentrated) dissolves the glycosides, a solution containing digitalin becoming golden yellow, and one containing digitonin becoming red. On heating, the solution containing digitonin becomes purplish or black. A solution of digitonin in 50 per cent. sulphuric acid (by volume) is yellow, and becomes red, then purple or black on heating. A solution of the mixed glucosides similarly treated may become a dingy green, progressing to black.

Sulphuric Acid and Bromine. A solution of digitalin in sulphuric acid, exposed to bromine vapour, becomes violet ; under similar circumstances, digitonin remains red, though the colour is intensified by bromine.

Keller's Test. The glycoside is dissolved in 1 cc. of glacial acetic acid containing 5 per cent. of ferric sulphate, and this solution is floated on the surface of a mixture of concentrated sulphuric acid (100 parts) and 5 per cent. ferric sulphate (1 part). Digitoxin gives a bluish-black ring at the zone of contact, and after about two hours the acetic caid layer (*i.e.*, the upper one) becomes blue. Digitalin gives an immediate cherry-red colour in the upper part of the sulphuric acid layer.

Lafon's Test. A small amount of the solid glycoside is moistened, first with a drop of concentrated sulphuric acid diluted with its own volume of 95 per cent. alcohol, and then with a drop of very dilute ferric chloride solution. Digitalin gives no colour, but digitoxin gives an intense greenish blue.

Baljet's Reaction. With picric acid and sodium hydroxide, digitalin gives a deep red colour ; gitalin, digitoxin, and strophanthin (as well as peptones, creatinine, acetone, and reducing sugars) give an orange red, digitonin, arbutin, and amygdalin do not react.

Fagge and Sir Thomas Stevenson found the physiological test applied to a frog the best test for the presence of digitalin,[2] and this is still true. The physiological test should always be used in confirmation of the chemical tests.

[1] Digitonin is a saponin, without therapeutic action on the heart.
[2] Guy's Hosp. Rep., 1866, p. 37.

Cases. Digitalin has acquired some notoriety by reason of the trial of Couty de la Pommerais, at Paris, in 1864, for the murder of his mistress, a woman named Pauw. The deceased, who was about forty years of age, and in the enjoyment of good health, was suddenly seized with violent vomiting, and, after an illness of about twenty-four hours, died on November 17th, 1863. The accused had induced her to insure her life in various insurance offices for enormous sums of money, quite disproportioned to her circumstances. Immediately after her death he put in a claim for these large insurances. The body of the deceased was exhumed, and inspected for the first time thirteen days after death. The viscera throughout were healthy; they presented no unusual appearance, and revealed no natural cause of sudden death. The stomach and bowels, which were well preserved, bore no marks of the action of poison; and, on a chemical analysis, no poison of any kind could be detected in these organs by Tardieu and Roussin. The symptoms, during the illness, owing to there being no suspicion of poisoning, were not accurately observed. Repeated vomiting, with great depression and exhaustion, seem to have been the most prominent. Failing to detect any poison by chemistry and the microscope, the experts adopted the physiological test of administering prepared alcoholic and aqueous extracts of the stomach and intestines to animals. An attempt made to separate the active principle and remove the organic matter by dialysis did not yield satisfactory results.[1] Seventy-five grains of the mixed extracts above mentioned were introduced subcutaneously into the thigh of a dog. The animal vomited twice; and in four hours the pulsations of the heart sank from 102 to 86; its action was irregular and intermittent, and the respiration was deep and painful. There were no narcotic symptoms; on the next day, the dog was better, and it completely recovered. Sixty grains of these extracts in water, administered to a rabbit by means of a funnel, caused death in a few minutes, probably from syncope (or asphyxia ?).

The deceased, during her fatal illness, had vomited on the floor of her room. An alcoholic extract was made of the scrapings of the floor and of the substances deposited between the planks. No mineral poison was found in it. Seventy-five grains of this extract were introduced subcutaneously into the thigh of a dog. The animal suffered from vomiting and depression of the action of the heart, and died in about twenty-two hours. There was no coma nor insensibility at any time. Thirty-one grains of the same extract diffused in water were administered to a rabbit by means of a funnel. In less than three hours after the injection the animal died, having suffered from irregular and depressed action of the heart. Sixty grains of an alcoholic extract from the scrapings of the floor, said to be free from vomited matters, had no effect upon an animal.

These two extracts of the floor had different chemical properties. The first, containing, as it was believed, a portion of the vomited matters, amounted to half an ounce. It was of a brown colour, had a rancid oily odour, and a bitter taste. Its solution was precipitated by tannic acid; it was coloured purple red by sulphuric, and green by hydrochloric acid. The second was coloured, had an oily aspect, but no bitterness. It was not precipitated by tannic acid, and was feebly coloured by sulphuric and hydrochloric acids; the results being different from those obtained with the first extract. It was objected to any inferences from the properties of these extracts that deceased's room had been formerly occupied by a photographic artist; but it is expressly stated that no noxious mineral substances such as are used in photography, were found in them. No attempt was made to procure digitalin from the extracts; the presence of this principle was a matter of inference, from the extracts produced; and the reason assigned for the extract derived from the stomach and bowels of deceased having no fatal effect upon animals, was that the quantity of the active principle left in the body at the time of death was too small.

Tardieu and Roussin deposed at the trial that the deceased had died from a vegetable poison which produced no marked change in the body, which could not be revealed by chemical analysis, but only by its noxious effects on animals. The effects on animals were in this case similar to those caused by digitalin, and without positively affirming that the deceased woman, Pauw, had died from this poison, there was the strongest presumption that she fell a victim to it. The deceased was quite well the day before her death, and the *post-mortem* examination of the body proved the absence of any natural cause to account for this sudden death.

[1] *Ann. d'Hyg.*, 1864, 2, 105.

In reference to the accused, it was proved that he had in his possession a large number of poisons of a deadly kind, including digitalin; that he had at three different times purchased as much as fifty-two grains of this poison, of which much had been used, and that those quantities were inconsistent with any reasonable medical requirements. As the accused was a homœpathic practitioner, the purchase and actual use of such large quantities of so potent a drug were quite inexplicable on any theory consistent with his innocence. On the other hand, the case was equally against him in its moral aspects: it was clearly established that, by reason of the large insurances effected on her life, he had a strong motive in the death of the woman, that a long cessation of their intimacy had taken place by reason of his marriage with another person, that he had suddenly and without any reasonable grounds renewed his intimacy with the deceased, and the date of her fatal illness was in accordance with these visits thus renewed. In short, De la Pommerais had the motive, means, and opportunity of destroying the life of this woman by poison, and no theory consistent with his innocence could be suggested, by those who defended him, to explain satisfactorily the mass of moral and medical circumstances which were clearly proved against him. Further, he overacted his part, and by forged letters and correspondence had shown that he had fully anticipated the sudden death of the woman Pauw, and the explanations that might be required of him in order to account for this event. Apart from any questions respecting the speculative character of the medical evidence, there were circumstances proved in this case which were inconsistent with any theory of the innocence of the accused. The jury found him guilty of murder, and he was executed.[1]

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Poisoning by Erythroxylon coca (Cocaine)

Source and Method of Occurrence. *Erythroxylon coca* is the plant from the leaves of which cocaine is extracted. It grows or is cultivated in most tropical countries. Coca leaves contain from 0·6 to 2 per cent. of total alkaloids. There are four or five alkaloids present, all of which are derivatives of ecgonine. Cocaine is used very largely as a local anæsthetic, with, in a good many cases, most disastrous results, both from acute poisoning and from the formation of the habit of taking the drug in small quantities. Synthetic substitutes (novocaine, β-eucaine, etc.) are very frequently employed as local anæsthetics and though less toxic than cocaine, have caused fatal accidents. The symptoms resemble those of cocaine poisoning. Mayer[2] has discussed in some detail the toxicity of cocaine and other local anæsthetics. Procaine appears to be the safest of these but the concentration should not exceed 1 per cent. Cocaine should not be injected but used for surface medication and the

[1] Tardieu, "Etude Médico-légale sur l'Empoisonnement."
[2] Mayer, E., *J.A.M.A.*, 82, 876 (1924).

total amount should not exceed 1½ grains. The patient should be recumbent if possible. Urethral injections appear to be specially dangerous and should be avoided if there has been any trauma.

The official preparation of the plant is the alkaloid, the dose of the hydrochloride of which is, officially, one-eighth to one-quarter of a grain. Lamellæ, containing one-fiftieth grain, trochisci, containing one-twentieth grain, and an ointment containing 0·25 per cent. are also prepared. Many preparations of coca leaves are made, elixir, extract, infusion, vinum, etc., none of which is official.

The homicidal use of cocaine is very rare, and only a few accidental and suicidal cases are reported.

The drug is probably the most popular of all for drug addicts, and its use appears to be increasing in many parts of the world.

Toxicity and Fatal Dose. The ordinary fatal dose of cocaine has been placed at about 18 grains but death has been reported from ⅓-grain. In regard to toxicity, the power of the resistance of the human frame seems to be capable of almost indefinite increase by habit (as in the case of opium). Mann records the case of a man who habitually injected twenty-three grains daily beneath his skin, and much larger habitual doses are on record. Sir Thomas Stevenson, in a former edition of this work, stated that even one-sixth of a grain, hypodermically, might cause toxic symptoms.

For a case where one grain injected for tooth extraction proved fatal, *vide Lancet*, 1901, 2, p. 533.

A case of recovery after eight to nine grains was reported in the *Lancet*, 1895, 1, 281.

A fatal case from swallowing two drachms of a 10 per cent. solution, equal to twelve grains approximately, is reported with autopsy in the *Lancet*, 1895, 2, p. 1104.

The first published fatal case in England was that of a man who was given in mistake twenty grains and succumbed in about an hour.[1] A fatal case occurred in Russia from a dose of twenty-two grains administered by rectal injection.[2] Recoveries have taken place after very large doses taken into the stomach. The most typical case of the kind was that of a man who swallowed forty-six grains and recovered.[3] One-twentieth of a grain given subcutaneously to a girl gave rise to dangerous symptoms, and a hundredth of a grain applied to the eye of a patient aged fourteen years occasioned symptoms of poisoning.[4]

For other cases, see *Lancet*, 1913, 1, 16.

Duration. Local anæsthesia is produced within a few minutes by cocaine solutions, and a similar period usually elapses before ill effects are produced by toxic doses in acute cases. As in the case of morphia, danger is not passed for many hours after a large dose taken by one unaccustomed to the drug.

Symptoms. The drug acts first as a stimulant to the highest centres of the brain, and the stimulus gradually descends to the lower centres, the motor area, the medulla and cord.

This stimulation is followed by depression in the same order, but the vital centres in the medulla appear to be the last to become paralysed.

[1] *Lancet*, February 9th, 1889.
[2] *Ibid.*, September 28th, 1889.
[3] Dixon Mann, *loc. cit.*
[4] Hamilton, "System of Legal Medicine," 1st ed., 1894, vol. 1, p. 429.

Small doses ($\frac{1}{4}$ to 1 grain) produce a genuine stimulation of the brain, and the person feels well, talks freely, and is often hilarious and happy. It is this feeling of well being and the abolition of fatigue which induces so many persons to continue taking the drug. With large doses (5 to 10 grains) the excitement is increased and the patient cannot keep still, the pupils are dilated, the pulse rapid (100 to 110), and the respirations increased. Deep sensibility is lost. Confusion of the mind follows, and there may be giddiness, fainting and nausea; the temperature may be raised two or three degrees. Convulsions are usually a prominent symptom and should be controlled by the use of barbiturates. Convulsions may be followed by collapse, and death takes place from paralysis of the respiratory centre.

When death takes place after use of the drug as a local anæsthetic, it is often due to too rapid absorption, for it has been demonstrated that the organism can tolerate relatively much larger doses when the drug is slowly absorbed.

A man, *æt.* 40, injected beneath his skin half a grain of the alkaloid to remedy the after-effects of drunkenness. Twenty minutes later he was found lying on a doorstep, pale, with dilated pupils, and the conjunctiva of the eyes insensitive. The breathing was slow and difficult, the pulse 140 in the minute. He was sensible, unable to articulate, and could not swallow, liquids being rejected from the mouth. The patient was in a serious state for some time, but recovered. This man had previously had repeated doses of half and six-tenths of a grain administered by injection every half-hour till three grains had been used, without ill effects.[1] In other cases alarming symptoms of depression have been produced by the application of solutions of cocaine to mucous membranes, and when injected before surgical and dental operations.[2]

Other symptoms which may be observed are: deafness; loss of taste and smell; profuse perspiration; intermittent pulse; shallow, irregular, gasping, convulsive breathing; impairment of gait and speech; muscular rigidity; convulsive twitchings and paralysis may occur. It markedly increases the sexual desire in both sexes, but in the male the increased desire is accompanied by a diminution in the physical powers which leads to sexual perversions.

In chronic cases it is difficult to state what symptom may arise. As in morphia addiction, the habit of taking cocaine seems to sap the moral fibre of its devotees, and in their efforts to obtain the drug, they become reckless of everything—lying, cheating, stealing—nothing comes amiss to them in their efforts to satisfy their craving. The general symptoms comprise digestive disorders, loss of appetite and emaciation. Restlessness, irritability, and sleeplessness are common. Hallucinations, delirium, tremor, convulsions and states of mental confusion may occur. The patient often complains of worms crawling under his skin. The pupils are usually dilated and the pulse weak and rapid. If the drug is taken as a snuff, the nose may be inflamed, and frequently an ulcer or perforation of the nasal septum may be found. The cases described below illustrate these points in some measure.

In tropical climates, *e.g.*, Bolivia and Peru, where coca leaves are chewed, it seems to make great exertion possible in cases where without the drug such would be impossible.

[1] *B.M.J.*, 1887, 1, p. 524.
[2] *Ibid.*, p. 676; 1888, 1, p. 151

Duration. The duration of intoxication may be very short, *i.e.*, half an hour or less; but it is sometimes prolonged. When death ensues from one dose it may take place within as short a time as twelve minutes, or may be delayed for several hours (*vide* cases on p. 701).

Treatment. In acute cases the stomach should be emptied and washed out with dilute permanganate solution. The mouth and nose should also be carefully washed out. A suspension of animal charcoal may be introduced into the stomach with the object of adsorbing any of the drug which remains on the mucous membrane. The bladder should be washed out. General stimulants such as ammonia, ether, etc., may be used. Nitrite of amyl is said also to be of assistance used as an inhalation. Strong coffee, strychnine, and digitalis may also be tried. Chloroform or a barbiturate may be used if spasms are violent. Artificial respiration may be necessary.

In chronic cases institutional treatment must be insisted upon if the patient can afford it. The drug should be withdrawn at once. The heart should be carefully watched, and the insomnia treated by bromides, veronal, etc. The gastric irritation must be carefully treated.

Post-mortem Appearances. There is nothing characteristic, but as the drug is so frequently taken hypodermically, marks of punctures of the needle should be looked for, while in addicts to the snuffing habit perforation of the nasal septum may be found.

Analysis. Cocaine may be sought for by the method on p. 265.

Delay should be avoided as far as possible, and the tendency of cocaine to hydrolyse (to methyl alcohol, benzoic acid, and ecgonine) must be remembered. Benzene is a useful solvent in the final purification.

To the alkaloidal residue left after the final evaporation of the organic solvent, the following tests may be applied :—

1. If a few drops of strong nitric acid are added to a small quantity of solid cocaine, or to one of its salts, and the mixture evaporated to dryness on a water bath, the residue gives, on stirring with a drop or two of strong solution of caustic potash in absolute alcohol, a distinct odour of ethyl benzoate, recalling that of peppermint, or citronella, or meadow-sweet. De Silva, who first described this test, considers it to be an extremely delicate one for cocaine, but the odour is scarcely distinctive enough to render the test by itself an absolutely reliable one.

2. If a few milligrams of cocaine are heated in a watch-glass with concentrated sulphuric acid over a by-pass flame, benzoic acid is liberated and can be collected as a sublimate on a larger watch-glass inverted over the first.

3. If a few drops of a 5 per cent. solution of chromic acid in water are added gradually to a solution of cocaine hydrochloride, each drop produces a precipitate which immediately re-dissolves. If now a small quantity of strong hydrochloric acid be added, a heavy yellow precipitate of cocaine chromate is produced. This reaction is stated by Metzei[1] to be peculiar to cocaine. (Many alkaloids give precipitates in *neutral* solution. Novocaine and β-eucaine give no precipitates.)

[1] *Pharm. Zeit.*, 34, 697.

4. A drop of potassium permanganate solution is allowed to dry on a microscope slide. A drop of half-saturated alum solution is placed on it, and a fragment of the alkaloid added. Rectangular violet-red crystals appear. Novocaine decolorises the permanganate solution and β-eucaine gives a violet oily precipitate.

5. Gold chloride solution, added to a cocaine solution, gives a precipitate which is amorphous at first but becomes crystalline after a short time or on the liquid being warmed and slowly cooled again. The crystals form delicate rosettes or resemble fern-fronds. Novocaine and β-eucaine give amorphous precipitates.

6. The physiological test may be used by applying a solution of cocaine hydrochloride to the tongue or lips, when a feeling of numbness is produced, due to the local anæsthetic action of the alkaloid.

7. When cocaine is heated with alcoholic potash, methyl benzoate is liberated and can be recognised by its odour.

8. Novocaine contains an amino-group and therefore gives the diazo reaction.

Cases. Percy Smith records [1] the case of a nurse, aged thirty-nine, who entered Bethlem Hospital as a voluntary boarder for the cocaine habit. She had previously been addicted to the use of morphine and laudanum, and her mother had been the victim of the morphine habit. She had commenced to take cocaine about eight months previously, and had gradually increased the dose until ten grains was her usual quantity, though she occasionally took twenty-four and even thirty-six grains at a single dose. For the first six hours after a dose of ten grains she felt more able and inclined for work whilst sitting, but she could not go about, as it produced a feeling of weakness ; at the end of that time she would be disinclined to do anything and would lie down, but could not sleep ; about a quarter of an hour after a dose she usually suffered from vertigo for an hour and from palpitation for some hours ; she also had great dryness of mouth, thirst and anorexia. After a large dose there was difficulty in swallowing. She had hallucinations, and imagined she saw people and heard them talking to her, and used to carry on conversations with them although she knew that they were hallucinations. The hallucinations soon disappeared after her admission, and after four months, including a stay at the country convalescent home, she left apparently well, and two months later reported herself as quite well.[2]

The following case is recorded[3] by Dr. Barratt :—

A medical man, aged about thirty, gave himself a hypodermic injection of cocaine to relieve local rectal pain. This not proving effectual he repeated the injection about five minutes afterwards, each time filling the syringe completely, the total amount given measuring forty minims. Immediately after the last injection he noticed that the solution used was a 35 per cent. solution, instead of being, as he had imagined, a 1 per cent. solution. He had by him solutions of the different strengths in bottles of the same size and shape, hence the mistake. The total amount of cocaine hydrochloride injected hypodermically was therefore about fourteen grains. Feeling weak, he lay down, and I saw him about eight minutes after the second injection. He was then perfectly conscious, with a moist skin, and almost pulseless at the wrist. An injection of five minims of ether was given hypodermically, together with some brandy and hot tea by the mouth. The effect of the ether was to cause the radial pulse to revive, but at the end of seven minutes it again became imperceptible. These ether injections were therefore repeated again and again every seven to ten minutes for nearly two and a half hours, being resumed as soon as the pulse became thready. Meantime the patient complained of thirst, drank copious draughts of hot weak tea, perspired very profusely, and passed large quantities of almost colourless urine. He was in a feeble

[1] *Jour. Mental Science*, July, 1892.
[2] *B.M.J.*, Epit., August, 1892, p. 21.
[3] *Ibid.*, 1896, 1, p. 1032.

apathetic state, but quite conscious, and there were no convulsions. The ether injections caused scarcely any sensation to pain, although sensibility to touch was apparently unimpaired. At the end of rather more than an hour the heart's action became intermittent, every third beat being missed. A change of posture was suggested, and this symptom passed off after a time. In all two drachms of ether were given hypodermically, and two ounces of brandy by the mouth, and eighty ounces of urine were passed in the course of five hours, the patient taking about the same amount of liquid by the mouth. At the end of three hours it was found possible to leave off the injections of ether, and at the end of five hours the pulse had to a large extent regained its tone, while the weakness was less marked.

Next day the patient stayed at home, and seemed fairly well except that he was dull and had little appetite. On the second day he walked out for about a quarter of a mile, and then became very faint. Otherwise his recovery was uninterrupted, and there were no after ill effects. The extreme collapse, the absence of convulsions and unconsciousness, and the conjunction of perspiration and polyuria, are the striking features of this case.

In July, 1904, a somewhat unusual case came before the courts, which was thus reported by *The Times*, July 11th, 1904 :—

At Gloucester, recently, Mr. Justice Lawrence and a special jury heard an action in which Mr. Albert Edward Freeman, a young man living at Stroud, and employed as a carpenter there, sued Mr. Ebenezer Apperly, dental surgeon, of that place, for damages in respect of negligence alleged to have been committed by the latter's son and assistant, Mr. Henry David Apperly, in the stopping of a tooth. The litigation was begun about a year ago, and had a remarkable history. The case was begun in the High Court, and, at the defendant's instance, was remitted, on the ground of the plaintiff's inadequacy of means, to the County Court. It was heard at Stroud, where the jury disagreed. It was re-tried at the Gloucester County Court, where his Honour the learned judge, at close of the plaintiff's case held that there was no evidence to go to the jury, and stopped the case. A Divisional Court set aside this decision, and ordered a new trial, giving the costs of the second trial and of the appeal to the plaintiff in any event and directing that at the defendant's option the trial should take place at Gloucester Assizes, with a special jury, costs, however, to be on the County Court scale. The facts, briefly stated, were these :—On Christmas Eve, December 24th, 1902, Mr. Apperly, jun., put a temporary stopping into a tooth of the plaintiff. The dressing consisted of oil of cloves, carbolic acid, carbolised resin, and cocaine lanoline. According to the defendant's case, not more than one twenty-fifth of a grain of cocaine lanoline was used. After leaving the surgery the plaintiff was dizzy and faint and staggered. The next day the stopping came out at dinner, and the plaintiff had afterwards a repetition of the symptoms. He was seriously ill for some six months.

For the plaintiff it was sought to be shown that the cause of the illness was cocaine poisoning. It was said that Mr. Apperly, jun., had inadvertently used a larger amount of the solution of cocaine lanoline than he thought, and alternatively, that he ought not to have used cocaine at all without the plaintiff's consent or the authority of a doctor. Cocaine was described as a dangerous and erratic drug, not unlikely to cause serious illness if administered, even in small quantities, to persons susceptible to its effects.

For the defendant a large body of expert evidence was given. Physicians and surgeons were called to testify that the history of the plaintiff's illness was inconsistent with the theory of cocaine poisoning; dentists were called who said that the treatment of the tooth was perfectly right, that the use of cocaine was recognised as correct by all experienced dentists, and that ill results rarely, if ever, followed from its use.

His lordship summed up and left to the jury two questions: Did the plaintiff suffer from cocaine poisoning? If yes, Was the fact of cocaine poisoning due to the negligence or unskilfulness or incompetence of the defendant's assistant?

The jury, without leaving the box, answered "No" to the first question, and they added that, even if the plaintiff had been proved to have suffered as alleged, they would have acquitted Mr. Apperly of all blame.

The learned judge entered judgment for the defendant accordingly.

The following information relating to this case, reported by Dr. A. B. Davies, of Stroud, is of some interest :—

" Last Christmas I was sent for to see a patient alleged to be dying. When I got there he was all right, but his friends informed me that he had had several attacks of unconsciousness lasting for a variable time. It appears that the day before he had had toothache and had the cavity of the tooth ' dressed.' A short time afterwards he swallowed the ' dressing,' and since then had felt ' queer ' and was under the impression that he had been poisoned. I made light of it, but two days later a certain amount of loss of sensation was felt in both legs, with increased knee jerks. Suspecting that the symptoms might be due to cocaine, I went to see the dentist, and found that he had used a little ' cocaine lanoline ' to the tooth. The patient gradually improved, but remained in a neurasthenic condition for months, and was quite unable to follow his occupation (carpenter) because any exertion brought on fainting attacks."

The following is worth attention from its likeness to strychnine poisoning[1] :—

" About 7.40 a.m. on October 7th, I was asked to see M.C., who was said to have swallowed some cocaine. On arriving a few minutes after, I found a doctor in attendance and the patient dead. Her parents made the following statement : M. C., aged sixteen years, rose about 6.30 ; after dressing, she went into her father's bedroom, and swallowed some cocaine from a vial on the dressing table to allay the toothache. She then went downstairs. She had just taken two mouthfuls of hot tea when she felt faint, and in trying to go upstairs, fell. She was then assisted upstairs, and sat down on a chair ; she then had a convulsion and fell on the floor. Her father coming in asked her what she had been taking, and she said that she had taken cocaine for the toothache. Immediately thereafter she had a series of six convulsive fits in succession, the arms and legs being most affected, the face least ; there was frothing from the mouth, towards the end blood-stained. She never regained consciousness, and at 7.30, when the first medical man arrived, he found no signs of life.

" It was about 6.50 a.m. when she swallowed the drug, so that death took place in forty minutes. The quantity she had taken was about two drachms of a 10 per cent. solution, equal to twelve grains of the salt. The large dose, the fact of its being taken the first thing in the morning on an empty stomach, and the hot tea taken immediately after, would all tend to quicken the effect. The medicine was contained in an ordinary clear glass vial, and the patient's father had procured it without any prescription. There was no *post-mortem* examination."

Poisoning by Eucalyptus and other Volatile Oils

These substances, obtained by distillation, consist mostly of terpenes or of terpenes combined with phenols, ketones, aldehydes, such as camphor, and thujon (from oil of absinthe), sabinol (from oil of savin), eucalyptol (from eucalyptus and cajaput), etc.

The properties of most of the essential oils are similar, although they have been described in different parts of the text. Many of them produce marked irritation of the gastro-intestinal tract, and the oils of tansy, savin and rue are particularly marked in this respect. They may also exert an irritating action on the kidney during excretion.

Eucalyptus, like turpentine, imparts an odour of violets to the urine. The action on the central nervous system varies considerably with the different oils. Absinthe and camphor (already described) may cause a marked and prolonged excitement. The other oils may produce a transient excitement, followed by depression, and the depression of the respiratory centre may cause death from asphyxia.

[1] *B.M.J.*, 1895, 2, p. 1162.

Tremors, ataxia, or convulsions may be observed, but generally the narcotic effect is more pronounced than the irritation. In fatal cases, especially after continued use, fatty degeneration of the liver and kidney may be found.

The following case of non-fatal poisoning from eucalyptus[1] may be of some interest :—

"A boy, aged sixteen, was recovering from measles when he was given by mistake half an ounce of eucalyptus oil. He swallowed the draught from the medicine glass without looking at it, and immediately said, 'You have given me eucalyptus instead of my medicine.' He felt some burning in the mouth and tried to cough the oil up, without success. In two or three minutes he suddenly became faint, and, according to the parents' account, was quite unconscious. On arrival ten minutes later I found the patient much collapsed, almost pulseless, and completely unconscious. The conjunctival reflex was nearly absent, and the breathing stertorous. With difficulty I got a little mustard and water down, but as there was no proper reflex action of swallowing I desisted. However, the boy vomited twice ; whether this was due to the poison or the mustard I cannot say. His colour and pulse improved, but as I was not satisfied I washed out the stomach three or four times with the stomach-tube. Three hours later the bowels acted involuntarily, but the patient remained comatose for another hour, when coughing commenced and the conjunctival reflex became brisk. Two hours later still speech and recognition of relatives had returned, and the subsequent recovery was uneventful. The patient told me that he remembered nothing of what happened from the time of his feeling faint at 11 a.m. until about 5 p.m. On recovery of consciousness he complained only of thirst ; there was no abdominal uneasiness. The symptoms therefore were those rather of a narcotic poison than of an irritant."

Other cases have been recorded in the *B.M.J.*, 1906, 1, 558, 1020, 1085 ; 1925, 1172 ; 1927, June 4th.

Analysis. The oil may be separated from stomach contents, etc., by distillation. The (usually opalescent) distillate is saturated with sodium chloride and extracted with ether. Evaporation of the ether yields the oil, which may be compared with samples of known oils as to odour, behaviour on exposure to light and air, refractive index, degree of unsaturation, etc.

Poisoning by Fungi (Genera and Sp. Var.) : Mushrooms

Method of Occurrence. There are many varieties of fungi edible and poisonous, and the toxic symptoms of the poisonous varieties vary with the species. Some of these cause characteristic effects on the central nervous system but in general there may be observed either alone or together with nervous effects, symptoms referable to the gastro-intestinal canal, *e.g.*, nausea, vomiting and violent abdominal pain often with bloody stools. Mushrooms contain a large quantity of water, and are very rich in albuminous constituents, so that they are prone to decomposition. They are also for some people indigestible, and apt to produce catarrh of the stomach and intestines and it appears that some individuals have a marked idiosyncrasy to mushrooms.

In the classical period mushrooms were largely used as articles of diet, and cases of poisoning were not uncommon. Euripides in a single day lost his wife, two sons and a daughter from this cause (Paulet). Of other famous people may also be mentioned the Emperor Jovian, the Emperor Charles VI., Pope Clement VII., and the widow of the Czar Alexis. Between 1749 and 1788 Paulet states that in the environs of

[1] *B.M.J.*, May 16th, 1925.

Paris there were over one hundred deaths from mushroom poisoning. Since his time many hundreds of cases have been chronicled, especially by French mycologists, and numerous cases have occurred in America, Italy, Germany and Japan (480 cases in eight years, Inoko). In 1900 Gillot collected the records of over 200 cases, chiefly in France, and in another seven years Ford collected as many more. In the vast majority of cases the poisoning has been accidental from mistaking poisonous varieties for harmless ones.

The most poisonous mushrooms belong to the family of Agaricineæ, and it is especially the genus Amanita which has been the principal poisonous agent. This includes the poisonous species *Amanita phalloides* and its varieties, *Amanita muscaria* (fly agaric). Other poisonous fungi include the false morels (Helvella) *Russula emetica*, *Boletus satanus* and *B. luridus*.

Amanita phalloides

A. phalloides, commonly called the "white" or deadly Amanita, and the allied species *Amanita verna* ("the destroying angel" of Bulliard), is the cause of the great majority of cases of poisoning by mushrooms. It is also known by a number of other names—*A. bulbosa*, *A. alba*, *A. citrina*, *A. mappa*, *A. venenosa*, etc.

It grows to a height of about six inches and consists at its base of an expanded cup (the poison cup), within which rests the stalk. The latter is surmounted by an expanded pileus, on the under-surface of which are the gills covered with white spores. The plant is pure white in colour with the exception of the pileus, which varies in shade to the colour of amber. It receives its specific name from the general resemblance to a phallus, although the analogy is nothing like so striking as in the case of *Phallus inpudicus*.

Toxicity and Fatal Dose. The toxicity is very high, but cannot be stated in definitely measured weight. Death may ensue from quite small quantities. Thus Plowright saw a fatal case in a child who had eaten one-third of the top of a small specimen. Cooking does not destroy the toxicity, the majority of the accidents having occurred with properly cooked material.

Symptoms. A prodromal stage of 6–15 hours in which there are no manifest symptoms ushers in an attack of extreme abdominal pain, accompanied by vomiting and diarrhœa, the vomit containing blood and mucus. Anuria is usually present, but hæmoglobinuria has not been witnessed. Severe cramp in the calves is fairly common. Remissions alternate with paroxysms of pain and vomiting, the suffering producing a characteristic expression which has been called by the French "la face vultueuse." Loss of strength, prostration, cyanosis, jaundice, and coldness of the skin supervene rapidly, and death with coma takes place within a few days. The prognosis is bad, the mortality being about 50 per cent.

Pathology of the Condition and Analysis. The active principle is a substance called phallodin which was isolated in 1937.[1] This substance

[1] Lynen and Wieland, *Liebegs Ann. d. Chem.* 533 : 100, (1937).

appears to have a toxic effect on the liver and kidneys, producing necrosis and fatty degeneration. It also produces in experimental animals a marked drop in blood sugar and a decrease in chlorides.

Portions of undigested fungus in the vomit are to be looked for: these would seem to be dislodged with difficulty, for they have been found as long as fourteen hours after ingestion.

Treatment. Little can be done except on general lines, as no drug has been found which acts as an antidote to Amanita toxin. The stomach and bowel must be washed out and a suspension of animal charcoal left in the stomach. Intravenous saline with glucose should be given to combat the collapse due to dehydration, and to replace the diminished glucose and chloride in the blood. Morphia may be required to assuage the pain.

Post-mortem Appearances. The stomach and intestines show intense inflammation or ulceration; there is usually intense fatty degeneration of the liver and fatty changes in the kidneys and heart. There may be general congestion of the viscera with hæmorrhages. Ford has succeeded in producing the majority of these lesions experimentally by the injection of Amanita toxin.

Cases. A boy, *æt.* 13, fried and ate for breakfast at 8.30 a.m. two fungi which he had found growing under a tree. He returned to his work without complaint. At noon he had his dinner of pork and vegetables. At 1 p.m. he returned to work, where he remained until 6 p.m., working the whole time without any complaint. Soon after he reached home, he complained of feeling ill, and vomited violently. Purging then followed, with severe spasmodic pain in the abdomen. These symptoms continued throughout the night until 6 a.m. The bowels then ceased to act. At 11.30 a.m. on the second day, he was suffering from constant pain in the bowels, occasionally aggravated; there was tenderness over the abdomen generally, but especially over the course of the transverse colon, with vomiting every ten minutes—great thirst, skin warm and perspiring, pulse ninety, and great depression. At 3 a.m. he was again seen. Vomiting and purging had returned. There was great exhaustion; pulse imperceptible; the action of the heart feeble. He was lying in bed on his back, with the knees drawn up. Sensibility and consciousness were perfect. He complained of great pain in the stomach; there was tenderness over the abdomen, but no distension. In another hour he died, *i.e.*, about forty-four hours after eating the fungi, and about thirty-four after the first setting-in of the symptoms. Others partook of the fungi, but in small quantity, and they did not suffer.[1]

In 1871, two children died from the effects produced by noxious fungi. Several other persons were placed in a precarious condition from the same cause. Some fowls died from eating portions of the mushrooms. Two children, a boy, *æt.* 8, and a girl, *æt.* 10, cooked some mushrooms for breakfast. The boy ate greedily of them, but permitted the girl to take only one mushroom. The symptoms produced in both children were similar, except that the boy had them in a severer and fatal form, and the girl recovered. Three of four hours after the meal, the girl was seized with violent pains in the head and abdomen. She vomited several times in the course of the day, was restless, thirsty, and had occasional muscular twitchings of the hands. The boy died twenty minutes after admission.[2]

A serious epidemic occurred in the Orphanage of St. Louis, near Pont-de-Maye, Gironde, in 1884. Large quantities of Amanita were gathered in ignorance and given to the children, of whom eleven died in five days. In 1885 Schröter reported an epidemic in Silesia of eleven cases with ten deaths.

[1] *Med.-Times and Gaz.*, 1863, 2, p. 536.
[2] Guy's Hosp. Rep., 1872, p. 228.

Amanita muscaria

Source and Method of Occurrence. This fungus, known as "fly agaric," from the use of its decoction as a fly poison, is more rarely the cause of poisoning than *A. phalloides*, due no doubt to the fact that it looks more likely to be inedible, the top of the pileus being of a beautiful yellow or red-brown colour. In consequence it is more rarely eaten. Even when consumed its poisonous properties are not so deadly, many of the victims recovering. Its active principle is an alkaloid Muscarine related to Choline. It stimulates the parasympathetic nerves.

The peasants of Siberia and Kamschatka prepare a liquor from *A. muscaria*, from which a violent intoxication ensues characterised by furious delirium. The poison is eliminated in the urine, which is sometimes drunk by other persons to produce the same drunkenness.

Symptoms. These appear within a very short time of the ingestion of the fungus. There is a marked flow of saliva followed by sweating and lachrymation, nausea, vomiting and diarrhœa. The pulse is slow and irregular (sometimes rapid) and the pupil contracted. The patient may become excited, there may be hallucinations of sight and hearing, and in many cases acute delirium and convulsions occur. In cases that recover the symptoms soon clear up.

Treatment consists of gastric lavage, with animal charcoal, intravenous saline with glucose and the use of atropine.

Cases. Prentiss has recorded the exceedingly interesting case of muscaria poisoning which befell Count de Vecchi, an attaché of the Italian Legation in Washington, and his physician. The Count purchased a quantity of mushrooms in Washington, and he and his physician ate them, remarking on their delicious flavour. Within fifteen minutes the Count was in a serious state, and within half an hour was completely prostrate and oppressed with the feeling of impending death. He developed blindness, trismus, difficulty in swallowing and was shaken by convulsions so terrific as to break the bed on which he had been placed. He died on the second day. The physician on returning to his office after the fateful breakfast became dizzy, and had diplopia and other ocular symptoms. He rapidly became unconscious and remained so for five hours, but recovered towards seven in the evening.

Amanita pantherina

The intoxication by this fungus is not so serious, for of thirty cases of poisoning collected by Gillot but two died. In Inoko's thirty-two cases one died. The fungus contains muscarine, the victims rapidly showing symptoms of drunkenness like that described above. The Japanese variety of this fungus causes remarkable nervous symptoms, including dilatation of the pupils, hallucinations, the sensation of insects crawling over the skin, visions of reptiles and beautifully coloured snakes, red, yellow, and brown. A jocund feeling expressed by laughing and singing was shown by all the patients. Recovery was usually rapid.

Morels and Helvellas

Helvellas are consumed in large quantities in France, Germany and Russia, and occasionally give rise to poisoning. The most important variety is *Helvella esculenta*, the active principle of which, Helvellic acid, is a hæmolytic substance, and produces hæmoglobinuria in dogs (Boehm and Kulz). If the fungus is boiled for a few minutes, the water strained off, and the fungus again rinsed, the whole of the toxic substances are removed. This precaution should never be neglected.

In man the symptoms are vomiting, diarrhœa, jaundice, hæmoglobinuria, delirium, convulsions, mydriasis, and coma.

Poisoning by *Russula emetica*, *Lactarius torminosus*, *Boletus satanas*, *Boletus luridus*, is also known, although rare.

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Poisoning by Gelsemium

Source and Method of Occurrence. The root of the plant *Gelsemium sempervirens* (known as yellow jasmine) contains two active principles, a crystallisable alkaloid, Gelsemine, which has a weak strychnine like action, and an amorphous substance, Gelseminine, which may actually be a mixture of alkaloids (the sempervirin, gelsemidin, and gelsemoidin of Sayre and Stevenson,[1] and the more recently isolated gelsemicine which is highly toxic to mammals.[2] : It is this mixture of alkaloids to which

[1] *J. Amer. Pharm. Assoc.*, 1915, 4, 60, 1458.
[2] Chou et Alia, *Chinese Jour. Physiology*, 5, 131 (1931)

the pharmacological effect of gelsemium is due. Gelsemicine has a latent period of 3-35 minutes after intravenous injection followed by respiratory arrest.[1] Another substance present is gelsemic acid, which gives a blue-green fluorescence to alkaline solutions and which also occurs in other plants. The extracts of gelsemium also contain peculiar resinous substances.

It is unfortunate that in certain German works and catalogues the name gelseminine (or " crystallised gelseminine ") is applied to gelsemine.

All the recorded cases of poisoning by gelsemium seem to have been accidental, and no case has yet given rise to a criminal charge.

Toxicity and Fatal Dose. Gelseminine has a very powerful depressant action on nerves, sometimes causing a tetanic condition and convulsions followed by death. It resembles coniine in its physiological action, but is more depressant to the central nervous system. It causes death by paralysis of the respiratory centre rather than by its action on the peripheral nerves. One drachm of the fluid extract is said to have caused death.

One-eighth of a grain by hypodermic injection killed a rabbit in one hour and a half. In fifteen minutes there were symptoms of great distress, and the animal was restless. In forty minutes there was great prostration, inability to move, gasping respiration, and the pupils were dilated, but there were no convulsions. From his experiments, Wormley infers that the quantity which proved fatal to the woman in his case could not have exceeded the sixth part of a grain.[2]

Gelsemine appears to have little action on mammals.

Duration. In the cases recorded below, death took place in four and seven and a half hours respectively.

Symptoms. Symptoms may be delayed for a considerable time, and toxic effects may be produced by small doses. Ptosis and double vision may first be complained of, followed by great depression, muscular weakness affecting the speech and gait. The respiration becomes slower and death is caused by respiratory failure.

Treatment. Empty the stomach as quickly as possible and leave in a suspension of animal charcoal. Atropine $\frac{1}{100}$ grain or strychnine $\frac{1}{30}$ grain may be given hypodermically in the hope of counteracting the depressant action of the drug, and nitrites (amyl, nitroglycerine, etc.) may be tried to relieve the heart failure.

Post-mortem Appearances. Nothing is to be found even suggestive of this form of poisoning without an exhaustive chemical analysis.

Analysis. In the Stas-Otto process, gelsemic acid is extracted by ether or chloroform from the acid solution, while the alkaloids remain in the aqueous layer and are extracted after the fluid has been made alkaline.

Tests for Gelsemic Acid. (1) Gelsemic acid crystallises in various shapes. (2) It is dissolved slowly by sulphuric acid, the solution varying in colour from yellow to a reddish brown according to the impurities

[1] Chen and Chou, *ibid*, 14, 319 : (1939).
[2] Chen et Alia, *Quart. Jour. Pharm. and Pharmacol.*, 11 : 84, (1938).
[3] *Amer. Jour. of Pharm.* January, 1879.

present; if a drop of ammonia solution is allowed to mix with a drop of this sulphuric acid solution on a microscope slide, gelsemic acid immediately separates in the form of crystalline needles. Wormley states that 0·001 grain gives a good precipitate, that æsculin does not give the precipitate, and that the test is not readily obscured by foreign matter. (3) Gelsemic acid dissolves in concentrated nitric acid to a yellow or orange-red solution, the colour depending upon the amount of substance. This colour changes, on addition of ammonia, to a deep blood red. The test is very delicate, but is also given by æsculin. (5) A solution of gelsemic acid in sodium or potassium hydroxide solution is yellow, with an intense greenish-blue fluorescence.

Gelsemic acid has been detected in the stomach contents some months after death.

Tests for Gelsemine and Gelseminine. (1) Gelsemine dissolves in concentrated nitric acid with little or no colour production (gelseminine gives a greenish solution), but if the solution is allowed to evaporate spontaneously, a bluish-green residue remains. (2) Gelsemine gives a red colour, changing to brown, with sulphuric acid. (3) With concentrated sulphuric acid and potassium bichromate (used as in testing for strychnine) gelsemine gives a red colour which changes, *viâ* purple, to a bluish green. (4) With vanado-sulphuric acid (Mandelin's reagent). gelsemine gives a red colour which fades to a pale violet. (5) Gelseminine gives a yellow colour with sulphuric acid alone, and on addition of potassium bichromate a violet colour changing to green.

Case. The following is of interest as a personal narrative. It is reported by Dr. Nankivell :—

"I took two ounces of the tincture of gelsemium instead of a glass of sherry, and returning to the dining-room, awaited the result. It was not long forthcoming. (We all live on the ground-floor here). The few feet travelled to the dispensary found me only too ready to accept the receipt of a helping arm, and in another minute the legs were paralysed. Dragging myself to the bedside with my forelimbs, they were unable to help me into the bed, into which I was lifted. There was no trouble so long as I lay quiet, but on the least exertion there were excessive tremors. Vomiting occurred during the next twenty-four hours. The temperature rose to 101·5° F. The heart's action was very violent and intermittent, possibly the aggravation of existing disease.

"All the muscles of the eyes must have been affected, but of all the voluntary muscles those of the right side suffered most. Prolonged conversation involved paralysis of the upper lip. The other symptoms were (1) somnolence, (2) no mental excitement, and (3) good appetite. The effect of the drug passed away as it began, from below upwards, but after the arms had recovered vision was not perfect for twenty-four hours."

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Poisoning by Helleborus niger (Christmas Rose)

Source and Method of Occurrence. This plant is not a native of England, though we have two, *H. viride* and *H. fœtidus*, which are probably just as noxious. According to Wibmer, the roots of the black hellebore (*Helleborus niger*) possess the greatest activity; but the leaves

are also highly poisonous when used in the form of infusion. By long boiling the poisonous properties of the plant are diminished. The roots and leaves have a local irritant action, producing in small doses violent vomiting and purging, with severe pain in the abdomen, followed by cold sweats, convulsions, insensibility and death. The experiments by Orfila on animals show that this poison acts like a *local* irritant when applied to a wound. Hellebore is a favourite quack remedy for worms, and has been given to procure abortion. It is not, therefore, surprising that it should be occasionally administered in an overdose, and cause death. The reader must, however, be careful to distinguish between these hellebores and the so-called green hellebore, or veratrum viride (*q.v.*)

Toxicity and Fatal Dose. The toxicity of the plant depends upon two active principles (glucosides): helleborin and helleborein. The former is a narcotic; the latter is a cardiac poison similar to digitalis, and is also a drastic purgative. The fatal dose is unknown, but half a drachm of a watery extract is recorded as fatal. The powdered root, in a dose of a few grains, acts as a drastic purgative.

Treatment. Evacuate the stomach, and counteract the collapse by stimulants, etc. No physiological antidote is yet available.

Post-mortem Appearances. Signs of irritation are likely to be present in the bowel and stomach.

Analysis. Helleborin, but not helleborein, may be shaken out of *acid* aqueous solution with ether (in which helleborein is insoluble); it is still more soluble in chloroform. After evaporation of the solvent the residue immediately yields a bright red colour on being touched with a glass rod which has been dipped in strong sulphuric acid. The red colour gradually disappears and a white powder separates.

Case. The following fatal case[1] is instructive in many ways:—

"A case of poisoning by hellebore occurring at Sackville College, East Grinstead, was recently investigated by the East Sussex coroner. A man took a quantity of powdered hellebore in mistake for liquorice powder. Twenty minutes later he was attacked with great pain. Mustard and water proved unavailing as an emetic, and all efforts to procure a medical man were fruitless. He remained conscious to within ten minutes of the end, and died within two hours, apparently from heart failure. The warden at the college stated that the effects of hellebore were that in the first place intense burning in the stomach ensued, and then, as absorption took place, it paralysed the nerve centre governing the heart, resulting, as in the present instance, in heart failure. Cases of poisoning by hellebore are undoubtedly rare. The symptoms are very similar to those produced by veratrum, and the two poisons may easily be mistaken for one another. Hellebore is strongly irritant to the mucous membranes, and has an action on the heart resembling that of digitalis, and it also acts on the nervous system. Hence in overdoses it produces nausea, vomiting, salivation, and diarrhœa. The primary stimulating action upon the motor centres and the heart may not appear, and the general depression will then be very marked. The heart's action becomes rapid and feeble, and death from syncope is likely to occur.

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[1] *Lancet*, 1, 42, 1904.

Poisoning by Hyssopus officinalis

Source and Method of Occurrence. This plant does not grow wild in Britain. The following is the only case of poisoning attributed to it.[1]

"A singular case lately formed the subject of a coroner's inquiry where a pregnant woman made an infusion of a pennyworth of hyssop, after taking which she died. She was stated to have been in bad health and the subject of Bright's disease. It is generally believed that the plants of the order to which this belongs—the Labiatæ—have no deleterious qualities, being carminatives and anti-spasmodics. However this may be as a rule, the medical witness at the inquest expressed his belief that the hyssop was the cause of the patient's death, and the jury adopted his opinion, returning a verdict accordingly."

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Poisoning by Ilex aquifolium (the Holly)

From some published facts, the berries of this tree appear to produce the effects of narcotic-irritant poisoning. A boy three years old ate a number of them. The symptoms which followed were sickness, pain in the head and abdomen, and much purging. Many of the berries of the common holly were passed in the motions; drowsiness supervened, and there was loss of consciousness. In twenty-four hours his face was pale: the skin pale and cool; pulse eighty, weak and small. The pupils were much contracted, but were sensitive to light. The vomiting had ceased, but there was some purging. Castor oil and stimulants were given, and on the second day the child recovered.[2] Wibmer speaks of these berries as having merely a purgative action.

Poisoning by Jatropha curcas (Physic Nut)

The *Jatropha curcas* is a West Indian plant which produces seeds containing an acrid oil, having some of the properties of croton oil. Four seeds act as a violent cathartic, and severe vomiting and purging have been produced by a few grains of the cake left after the expression of the fixed oil from the bruised seeds. The oil operates powerfully in a dose of from twelve to fifteen drops. It produces a burning sensation in the throat, vomiting, purging, and other symptoms of irritation, followed by inflammation of the stomach and bowels. One hundred and thirty-nine children in Dublin suffered from the effects of these seeds[3]; and in 1864 a number of boys at Birmingham suffered severely from eating some of them which they had found in a drug store, but they all recovered. Chevallier refers to a case in which thirty-three persons were poisoned by eating physic nuts. The symptoms from which they suffered were nausea, vomiting, and general depression. Twenty were so ill that they were placed in the beds of a hospital; the remaining thirteen soon recovered. The albumen of the seeds is said to have a flavour resembling that of the almond.[4]

[1] *Lancet*, 1, 124, 1899.
[2] *Lancet*, 1870, 1, p. 573.
[3] *Med. Times and Gaz.*, 1858, 2, p. 143.
[4] *Ann. d'Hyg.*, 1871, 1, 408.

The *Jatropha urens*, also a West Indian plant, is said to produce serious effects upon those who touch its leaves, which are covered with stinging hairs like those of the nettle. The wrist of a person accidentally came in contact with some of the hairs. After a few minutes there was swelling of the lips, redness of the face, faintness, great prostration of strength, and such a degree of collapse that for some minutes the sufferer was thought to be dead. He then rallied ; there was sickness, and within twenty minutes the man recovered. In another case the pain and swelling in the part touched lasted for some days, and an itching sensation continued for a longer period.[1] Assuming this account of the symptoms to be correct, the poison connected with the hairs not only has a local action, but it is very rapidly absorbed, and produces effects resembling those of serpent poisons.

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Richardière et Dalché, *Soc. de Méd.-lég. de France Bull.*, 1881–2, 7, 275.

Poisoning by Juniperus sabina (Savin)

Source and Method of Occurrence. This is a well-known plant (but is not a native of Britain), the leaves of which contain a poison in the form of an acrid volatile oil of a remarkable odour. They exert an irritant action, both in the state of infusion and powder. They yield by distillation a light yellow oil, on which the irritant properties of the plant depend. Savin is not often taken as a poison for the specific purpose of destroying life, but this is occasionally an indirect result of its use as a means of procuring abortion.

Symptoms. It causes violent pain in the abdomen, vomiting, colic, and diarrhœa. Urination is painful and the urine may contain blood. The uterus is congested and the menstrual flow increased, abortion sometimes occurs. There may be convulsions, coma and death either in a few hours or after a few days.

After death the gullet, stomach, intestines, and kidneys have been found either much inflamed or congested and there may be cloudy swelling or fatty change in the liver and kidneys. It has probably little direct action as an abortive, but, like other volatile oils it causes pelvic congestion and may by reflex irritation cause the uterus to expel its contents. Such a result can never be obtained without placing in jeopardy the life of the patient. On the other hand, a female may be killed by the poison without abortion ensuing. In 1845, Taylor met with a case in which death had been caused by savin powder, abortion having first taken place. Eight ounces of green liquid were found in the stomach, which, with the gullet and the small intestines, was highly inflamed. The poison was easily identified by the minute portions of the leaves found in the stomach. The *oil of savin* is also powerfully irritant[2], six drops

[1] *Pharm. Jour.*, April 17th, 1872, p. 863.

[2] Kagaza, Y., *Arch. f. exper. Path. u. Pharmakol.*, 124 : 245 (1927).

having caused severe toxic effects. For an account of this, see "Criminal Abortion," pp. 117, *et seq.*

Analysis. *See* under Eucalyptus.

Poisoning by Ligustrum vulgare (Privet)

Source and Method of Occurrence. The privet is not commonly classified among vegetable poisons, but the *berries* appear to exert a poisonous action. In 1853, three children ate the berries of the privet; two of them, a boy of three years of age and a girl of six, ate them rather freely. They suffered from violent purging, and when seen by a medical man the boy was found pulseless and cold, and before death he was frequently and violently convulsed. The girl was in a state of collapse, but rallied a little under treatment. Soon afterwards she died convulsed. The surviving child, who had only tasted the berries, did not suffer, and she was enabled to point out the shrub the berries of which they had gathered. In 1866, a child, *æt.* 2, died thirty-seven days after eating these berries, symptoms of irritation continuing more or less throughout. According to Loudon, they are eaten by birds when other sources of food fail.

In May, 1872, two children, aged twelve and eight years respectively, ate a quantity of leaves and shoots proved subsequently to have been those of the privet. The symptoms in both cases were drowsiness, convulsive twitchings, difficulty in moving about, loss of muscular power, severe vomiting and purging, the evacuations being of a greenish colour. They both recovered.

Cases. The following is from the *Lancet*, 1898, 1, p. 665:—

"In 1857, thirty-seven children suffered from poisonous symptoms after eating freely of acorns and privet berries. The symptoms were shrivelled appearance of the hands and face, cyanosis, intense thirst, and sickness; opisthotonos was a marked symptom in each case. All the children recovered.

"On February 18th, 1898, an inquest was held at York Town on the body of a female child, aged eight years, who died two days previously after a few hours' illness. On the 16th she complained of pain in the head and stomach. At dinner-time she seemed better, but whilst food was being prepared she gave a cry and became unconscious, and death took place before the arrival of a medical man. The child's teeth were tightly closed, her tongue protruding and her hands clenched. At the *post-mortem* examination the heart, liver, and kidneys were found to be quite healthy. The lungs were congested. The stomach was also much congested, with one patch of superficial ulceration about the size of a shilling. On inquiry it was found that the child had eaten privet berries. None of those were found in the stomach, but they had probably been discharged by vomiting. The symptoms were in accord with the previously reported cases to which we have referred, and we cordially commend the coroner's remarks that he hoped the evidence as to the privet berries would be a caution to parents as to their danger."

Poisoning by Lobelia inflata (Indian Tobacco)

Source and Method of Occurrence. This plant is imported from North America. Its leaves contain an alkaloid, lobeline, which is capable of producing poisonous effects on the brain and spinal cord, especially on the respiratory centre of the medulla, and resembles nicotine very closely in its action. The plant produces irritation of the stomach and bowels. When administered in doses of from ten to twenty grains, lobelia acts as an emetic; but in larger quantity it acts deleteriously,

and in smaller doses has an expectorant and antispasmodic action somewhat similar to that of tobacco. It has been proposed for the control of the tobacco habit, though toxic effects may be produced by its use.[1,2] It would also appear that even ordinary medicinal doses affect some persons with great severity.

There have been many inquests and trials for manslaughter in this country as the result of the improper administration of the powdered leaves of the *Lobelia inflata* by quacks and dealers in vegetable medicines. The medical evidence given at these trials has proved that in large doses lobelia is a noxious drug.[3]

In 1856, one of these quacks was convicted on a charge of manslaughter for causing the death of a woman by overdoses of lobelia. Severe pain, followed by loss of consciousness and congestion of the brain, were the chief symptoms preceding death in this case. The admission that, in proper doses, it was a useful remedy in spasmodic asthma, was of no avail on this occasion. The man was sentenced to three months' imprisonment.[4]

Toxicity and Fatal Dose. The toxicity of the plant would appear to depend on the alkaloid (lobeline) contained in it. There are no facts to determine the exact fatal dose of this, but in a case reported, a drachm of the powdered leaves proved fatal.

Duration. From a few minutes to thirty-six hours seems to be the variation in the total duration of fatal cases. As with other irritants, the effects on the stomach appear within a few minutes of swallowing the drug, but the rapidity with which the effects upon the nervous system appear would seem to depend upon the state and contents of the stomach at the time of taking the poison.

Symptoms. These would appear to be variable, probably according to whether the main effects were local on the stomach or whether they were exerted after absorption. There is usually a burning pain in the stomach, followed by nausea, salivation and vomiting. There may be muscular weakness, giddiness and inco-ordination. Clonic convulsions may occur. The pulse is slow at first. Severe collapse may occur with Cheyne Stokes respiration. Death may occur from paralysis of the respiratory centre, but although the symptoms may be alarming, recovery is the rule.

Treatment. This must be upon general principles, *e.g.*, stomach and bowel lavage, the use of animal charcoal for adsorption, attention to the state of the heart and respiration.

Post-mortem Appearances. Beyond the presence of portions of the plant which might be identified, it is not to be expected that anything will be found in any way characteristic of lobelia, though the appearance of the stomach will possibly suggest irritant poisoning.

[1] Dorsey, *Ann. Int. Med.* 1936, *10*, 628.

[2] Wright and Littauer, *J. Amer. Med. Assoc.* 1937, *109*, 649.

[3] See *Med. Gaz.*, vol. 44, pp. 383 and 384; vol. 46, p. 384; *Lancet*, 1853, 1, p. 237; *Pharm. Jour.*, August, 1851, p. 87; and for some remarks on the action of the poison see a paper by Curtis and Pearson, *Med. Gaz.*, 1850, vol. 46, p. 285.

[4] *R.* v. *Boyden* or *Jackson*, Lincoln Sum. Ass., 1856.

Analysis. Lobelia is seen in the form of a greenish-coloured powder (fragments of leaves). This powder acquires a reddish-brown colour with strong nitric acid, and is blackened by concentrated sulphuric acid. Iodine water has no effect upon the infusion. The ferrous and ferric sulphates produce with it a dark green colour, the ferric sulphate very rapidly.

The leaves of lobelia are generally seen in fragments which do not readily admit of identification by the microscope. The *seeds* are very small, of a lengthened oval shape, reticulated on the surface with projecting hairs or fibres, and of a light brown colour. Their presence among the fragments of leaves should furnish a sufficient proof of the presence of lobelia.

The leaves and seeds contain the alkaloid, which may be sought for by the processes described on p. 265, and when it has been obtained in comparative purity, the following corroborative tests may be applied :—

1. Strong sulphuric acid produces a red colour with the mixed lobelia alkaloids. Pure lobeline, which is official in the German Pharmacopœia (1926), is there described as giving no colour with sulphuric acid but a red colour with a formalin-sulphuric acid reagent (Marquis' Reagent).

2. Impure lobeline gives with sulphomolybdic acid a violet colour, which appears after about two minutes, increases in intensity, and finally fades to brown or yellow. Pure lobeline gives a brown and then an intense green colour. A similar colour is given by morphine, but morphine is a solid substance, the mixed lobelia alkaloids form a liquid ; moreover, morphine does not give a red coloration with strong sulphuric acid, as lobeline does (Luff).

3. Lobeline solutions are precipitated by tannic acid.

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Edmunds. *Jour. Pharmacol.*, 1910, 1, 27.
Johnson, *Med. Press and Circ.*, 1884, 37, 127.
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Tidy, " Trial on a Charge of Manslaughter for Poisoning by *L. inflata*." *Lond. Med. Gaz.*, 1850, 11, 384.

Poisoning by Lolium temulentum (Bearded Darnel)

Symptoms and Effects. Poisoning by darnel is generally the result of accident from the intermixture of the seeds of this grass with wheat or rye. The seeds are ground into flour and eaten with the bread.

It is now considered that the symptoms formerly ascribed to lolium, namely giddiness, tremor of the muscles and stupor, were in reality due to a fungus which had attacked the grass.

REFERENCE

Freeman, " The Seed Fungus of *Lolium temulentum*, L., the Darnel." *Phil. Trans. of Roy. Soc. Lond.*, 1904, 196, 1-27.

Poisoning by Manchineale Tree

Source and Method of Occurrence. The following case, reported by Dr. Caddy, is the only one to be found. The plant apparently grows

in the Grenadine Islands. The smell of the apple-like fruit is delicious, and has been the cause of many a sailor in these regions meeting his death from eating it.

"On June 2nd, 1894, a black child, aged five years, was admitted under the following circumstances: The mother stated that the child had been playing with some other children on the previous evening by the seashore, when she suddenly came running home crying violently, and complaining of severe pains in her 'mouth and belly.' She was very sick, but could not swallow food or water, and passed a very bad night, screaming loudly at times, and being extremely restless. Early the following morning the mother brought the child to the hospital, and upon my questioning her said that the child had been poisoned from eating some manchineale from one of the trees under which she had been playing. I found the child in a state of collapse, the surface of the body being bathed in a cold sweat, the radial pulse imperceptible, heart sounds hardly distinguishable, respirations thirty and very shallow, temperature 96·2° F. The lips were much swollen and covered with blisters, and the tongue was enormously swollen and blistered, interspersed with white eroded patches. There were also blisters in the palm of the right hand, and two small ones on the left. On palpation the abdomen was very tender, this being much exaggerated over the epigastric region. The pupils were widely dilated.

"I at once ordered the child to be placed in bed and surrounded by hot-water bottles, and injected subcutaneously ether sulph. R xxv. The child gradually rallied, and the after-treatment consisted in washing out the mouth with glycer. boracis, and the administration of the following mixture: R tinct. opii, ℥ x.; potass. brom. ʒss.; potass. chlor. gr. xx.: sod. bicarb. ʒj.; bismuth, subnit. ʒj.; mucilag. acaciæ ʒj.; aquæ ad ℥ij.: ʒj. every two hours, to be allowed to trickle down the throat a few drops at a time. An enema consisting of brandy ℥j., starch ℥ij., and beef-tea ℥ij. was given, but as this appeared to distress the patient, it was discontinued, and 'zyminised nutrient suppositories' every four hours substituted. On June 4th the temperature rose to 101° F., and as the patient was unable to swallow even a few drops of water, and ice could not be procured, the mixture had to be discontinued, and hypodermic injections of morphine sulph. gr. $\frac{1}{16}$ were given every five hours, while, to cope with the intense thirst, an enema of cold water was given every two hours, which appeared to cause great relief to the patient. The urine was excreted very copiously, and contained a trace of albumen. On June 6th the patient had decidedly improved, and continued to do so up to June 20th, when she was discharged well."

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Caddy, "Poisoning by Manchineal." *Lancet*, 1894, 2, 1478.
Gazeau and Reboul, "Note sur Action du Mancenillier." *Arch. de Méd. Nav.*, 1893, 9, 101.

Poisoning by Mentha pulegium (Pennyroyal)

Source and Method of Occurrence. This plant, though a native of Britain, is not common nor easy to find. It enjoys a vulgar reputation as a means of procuring abortion (*vide* p. 119), and it is only owing to its slight toxic properties that cases of poisoning by it are not more frequent. This, like so many other volatile oils is liable to cause irritant effects in the abdominal canal and in the kidneys, and may reflexly affect the uterus. Fatty degeneration of the liver and other organs may result from large doses.

Case. A woman was admitted to the parish infirmary, Liverpool, in an almost collapsed condition, suffering from symptoms of acute gastritis. She stated that vomiting began four days previously after she had taken a tablespoonful of pennyroyal. The excessive vomiting continued despite the usual remedies, but ultimately ceased under the influence of morphia and rectal alimentation. The patient, however, gradually sank, and died on March 19th.

"*Post mortem.* The stomach was extremely congested, especially towards the cardiac end, the small intestines showed thickening of their coat and intense con-

gestion, most marked in the lower part of the ileum. The large intestines were congested, but not to the same degree as the stomach or ileum. The uterus was normal in size, and there was nothing noticeable in the other organs except some congestion of the brain. At the inquest evidence corroborative of the woman's statement was given ; but as the druggist who had sold the pennyroyal said that in thirty years' experience he had never heard of a case of poisoning by this drug, the jury returned a verdict that ' death was due to gastro-enteritis set up by some irritant poison,' but did not decide what the poison was." [1]

Other cases are reported :—

Girling, *B.M.J.*, 1887, 1, 1214.
Napier Jones, *B.M.J.*, 1890, 1, 661, and 1913, 2, 746.
Macht, *J. Amer. Med. Assoc.*, 1913, 61, 105.

Poisoning by Myristica fragrans (Nutmeg)

Source and Method of Occurrence. The domestic nutmeg has a certain reputation as an abortifacient if taken in sufficient quantities, as the following case, reported by G. E. Reading,[2] would show.

A woman three months pregnant, in order to procure abortion, swallowed three powdered nutmegs. She was well till three hours after, when she vomited several times, and passed into a condition of low, muttering delirium, with occasional silly laughter, and hallucinations of a ridiculous character. She could be aroused from this by shaking, but would relapse almost immediately. There was also a strong sense of impending death. The pulse was strong and rapid. Twenty grains of chloral hydrate were given, which lessened the delirium and allowed the patient to obtain sleep. The delirium continued to recur, however, at intervals for the next twenty-four hours, during which grain doses of calomel were given every hour ; the next day the patient was quite rational. Abortion did not take place. The general symptoms of poisoning strongly recall those which appear in some cases of poisoning by cannabis indica.[3]

Simpson reports the following case :—[4]

" I found the patient lying upon the bed in a drowsy condition and very delirious, the delirium taking the form of confusion and mistaking one person for another. There were lucid intervals. She complained of a sensation of tightness across the chest, of vertigo and faintness upon attempting to stand. She had vomited several times. The pulse was seventy-five per minute and rather feeble. The pupils were normal. Inquiries elucidated the fact that the patient had, being a week over her menstrual period, taken two nutmegs, bruised, in a small quantity of gin. In the afternoon her condition improved. The next day she was very much better, but still had some vertigo and was very weak."

A case is reported [5] in which a woman took one nutmeg ground up in a glass of stout with the object of procuring an abortion. In four hours she became so giddy that she went to bed. Intense headache and abdominal pain came on. When seen by a doctor she was restless and excited, the face flushed, the pulse accelerated and the abdomen tender. The symptoms continued for thirty-six hours.

Poisoning by Nicotiana (sp. var.) Tobacco

Source and Method of Occurrence. Tobacco is the leaf of a plant belonging to the natural order Solanaceæ. It contains nicotine as the active principle. Snuff is tobacco ground to powder. Powdered tobacco is used to a certain extent in animal therapeutics, and extract of tobacco and preparations of nicotine are largely used in agricultural and horticultural work, for fumigating and spraying, as insecticides, sheep dips, worm powders and so on. Some of these preparations contain high

[1] *Lancet*, 1897, 1, 1022.
[2] *Therap. Gaz.*, September, 1892.
[3] *B.M.J.*, Epit., December 10th, 1892.
[4] *Lancet*, 1895, p. 150.
[5] *B.M.J.*, April 29th, 1911.

concentrations of nicotine, and are very dangerous. Tobacco has been used homicidally, but is far more important as a cause of accidental death chiefly by the ingestion of nicotine insecticides. Serious outbreaks have been caused by inhaled tobacco dust and in connection with nicotine extraction. Tobacco poultices applied to wounds are very dangerous.

A case of suicide from drinking a plant spray containing nicotine was reported in the public press on May 24th, 1931. A girl took a mouthful of a nicotine spray and died a few moments afterwards.

It has been used to aid the purposes of robbers ; and there is reason to suppose that porter and other liquids sold in brothels are sometimes drugged either with tobacco or with snuff prepared from it. In 1891 Sir Thomas Stevenson investigated a fatal case of poisoning by snuff which appeared to have been given in beer to a drunken man, by way of a joke. He died within about half an hour of its administration.

Many fatal cases are known from the use of tobacco enemata. The use of this form of enemata (against intestinal parasites) is now happily superseded by other remedies.[1] Gill[2] reports a case of poisoning from the use of a nicotine suppository.

Toxicity and Fatal Dose. While tobacco itself cannot be called very poisonous, the alkaloid nicotine is a deadly poison, and, like prussic acid, it destroys life in small doses with great rapidity. Two drops placed in the mouth of a dog will kill it. The fatal dose for man is placed at about one grain of nicotine or about 30 grains of tobacco. It is stated that there is sufficient nicotine in one cigar to kill two people, if it were injected into the circulation. A case of poisoning by this alkaloid which occurred in Belgium in 1851 was the subject of a trial for murder[3] :—

> The Count and Countess Bocarmé were charged with the murder of the Countess's brother, a M. Fougnies, by administering to him nicotine while he was dining with them at the château of Bitremont. The poison was forcibly administered. The deceased did not survive more than five minutes, and he was not seen living by any of the attendants. The possession of the poison, as well as the moral evidence, fixed the crime on the Count, and he was condemned and executed.
>
> The appearances after death were to a great extent altered or destroyed by the pouring of some strong acetic acid into the mouth and over the body of the deceased, in order to conceal or remove the odour of nicotine. Stas detected the poison in small quantity in the tongue, throat, stomach, liver, and lungs of the deceased, as well as in a wooden plank of the floor near to which he was sitting ; and it was this case which led him to devise his well-known process for the isolation of the alkaloids.
>
> A gentleman swallowed a quantity of nicotine from a bottle, and almost immediately afterwards was seen in the act of falling to the floor. He was carried to an adjoining room, but before this could be reached he was dead. The symptoms noticed were that the deceased stared wildly ; there were no convulsions, and he died quietly, heaving a deep sigh in expiring. Nicotine was detected in the stomach and in the liver and lungs, which, however, had come into contact with the stomach.[4]
>
> The quantity of nicotine taken could not be determined. The deceased appears to have been rendered insensible immediately, and to have died within three or five minutes after having taken the poison.

Goodman and Gilman suggest that the lethal dose for men is about 60 mg., although as little as 4 mg. may produce alarming symptoms.[5]

[1] See Cases, pp. 295, 835.
[2] *B.M.J.*, 1901, 1, 1544.
[3] *Ann. d'Hyg.*, 1851, 2, pp. 147, 167.
[4] Guy's Hosp. Rep., 1858, p. 354.
[5] *Pharmacological Basis of Therapeutics*, 1941, p. 491.

Symptoms. Nearly everyone is familiar with the depression and faintness, collapse, cold sweats, pallor, and rapid onset of vomiting which smoking produces in one unaccustomed to it. It is probable that the symptoms in the young smoker are not entirely due to the action of nicotine, but also to products of imperfectly burnt tobacco, such as carbon monoxide, and various pyridine derivatives.

When nicotine or a decoction of tobacco leaves is taken the above symptoms occur, but are more strongly marked, and commonly end in death. There is a hot taste in the mouth passing to the abdomen, followed by salivation, vomiting and purging. The breathing is quick and laboured. The pupil is first contracted, afterwards dilated. The pulse is usually slow. There is muscular weakness and inco-ordination, followed rapidly by collapse and death with or without convulsions. Large doses may cause death in a few seconds apparently from paralysis of the vital centres.

Death occurs usually within a few minutes after ingestion. We have notes of a case in which a man was seen to go into a shed in his garden : and exactly five minutes afterwards was found dead. Beside him was a bottle containing a dark fluid consisting of 95 per cent nicotine. The stomach contained 13 grains of nicotine. In subacute industrial nicotine poisoning, slowing and irregularity of the heart are the predominant symptoms, with salivation, sweating, nausea and vomiting. Angiospasms and muscular tremors may also be found.

A woman applied some leaves of tobacco to ulcers upon her legs. After some hours she suffered from sickness, dimness of vision, cramp in the legs, and great prostration ; she also complained of a numb feeling. On the third day there was great sleepiness, with headache and an irregular action of the heart. In about a week, she recovered her usual health.[1]

A case is reported[2] of toxic symptoms produced by application to the skin of the juice from an old pipe.

Namias relates an instance of a smuggler being poisoned by having covered his skin with tobacco leaves, with a view to defrauding the revenue. The leaves, moistened by perspiration, produced all the effects of poisoning. The pulse was small and feeble ; there was faintness attended with cold sweats. The operation of the poison seemed to be principally on the heart. In persons who have smoked tobacco excessively the action of the heart is frequently deranged—tachycardia, intermitteney, etc.

The "tobacco heart" of the heavy smoker is now well known to physicians. There may be irregularity of the heart, with extra systoles and occasionally attacks of pain suggesting angina pectoris. In heavy smokers a form of blindness is now known to occur. There may be narrowing of the field of vision and dullness in vision.

There is evidence to suggest that the continued ingestion of nicotine has a deleterious affect on fertility.[3]

Post-mortem Appearances. There is nothing of importance to be observed, but one may expect signs of irritation and the characteristic smell of tobacco.

[1] *Lancet*, 1871, 2, p. 663.
[2] *B.M.J.*, April 24th, 1926.
[3] *Jour Pharmacol*, 87 : 1 (1946).

Analysis. Nicotine may be separated from stomach contents, tissues, etc., by the Stas-Otto process. An alternative method consists in digesting the material with dilute acetic acid. The filtered extract is treated with lead acetate in excess, and the filtrate from this is freed from lead by means of hydrogen sulphide. The clear fluid is then made alkaline with sodium hydroxide, filtered if necessary, and submitted to steam distillation. The distillate, in presence of nicotine, exhibits the odour and chemical reactions of that alkaloid.

Nicotine is not the only alkaloid present in tobacco, but is the only one of toxicological importance. In the plant it exists combined with various acids—malic, citric, etc. The amount in ordinary tobaccos varies from about 0·6 per cent. to as much as 8 per cent., but Macedonian tobaccos can be obtained with a nicotine content of under 0·5 per cent. No general rules can be laid down as to the relative nicotine content of cigarette, cigar, and pipe tobaccos, nor do " strong " tobaccos necessarily contain more nicotine than " mild " ones.

Nicotine is a colourless liquid which rapidly turns brown on exposure to air. It has little odour when pure, but soon acquires the characteristic smell of tobacco when exposed to air or dissolved in water. It has sp. gr. 1·048, boiling point 246·7° C. (at 745 mm. pressure), and is lævo-rotatory, its specific rotation being given by Jephcott (*J.C.S.*, 1919, 104, 8) as $[\alpha]_D^{20} = -168{\cdot}56$. Its salts are dextro-rotatory and usually crystallise with difficulty. The free alkaloid readily dissolves in water, alcohol, ether, or chloroform. The aqueous solution is strongly alkaline. In spite of its high boiling point, nicotine resembles coniine in volatising at ordinary temperatures (so that the greasy stain it gives to paper tends to disappear), and is volatile in steam.

Tests. (1) Nicotine is precipitated by the usual reagents for alkaloids, and from solutions more dilute than those required in the case of the most important other liquid alkaloid (coniine). Thus platinum chloride gives a yellow crystalline precipitate of nicotine platinichloride at a dilution of 1 : 5,000 (coniine 1 : :1,000). The precipitates with mercuric chloride (white) and silver arsenio-nitrate (yellow) resemble those given by ammonia. Ammonia, however, gives no precipitate with iodine solution (nicotine gives a reddish-brown precipitate), tannic acid, or potassium mercuric iodide[1] (both of which give precipitates with nicotine).

(2) With mercuric chloride, nicotine gives a white *crystalline* precipitate soluble in dilute hydrochloric acid or acetic acid. Strychnine also gives a crystalline precipitate which, however, is almost insoluble in acetic acid ; most alkaloids (including coniine) gives amorphous precipitates. Ammonia must be removed before applying the test.

(3) Sulphuric acid and potassium bichromate give a green colour (chromium sulphate).

(4) (Tunmann's Test). A drop or two of an aqueous solution of nicotine are added to a drop of a solution of *p*-dimethylamino-benzaldehyde in fuming hydrochloric acid. A pink, and then violet colour is formed at the line of contour, and a reddish-violet colour spreads through the liquid. This colour increases in intensity and remains for some hours. Coniine does not give the reaction, and neither does pyridine.

[1] Except in relatively high concentration.

The test is even more specific if a trace of the alkaloid is treated with a 1 per cent. solution of *p*-dimethyl amino-benzaldehyde in sulphuric acid (40 ml. concentrated acid diluted to 100 ml. with water). A bright pink colour appears in the cold and can be discharged by the addition of either concentrated sulphuric acid or water.

(5) (Schindelmeiser's Test). Nicotine, free from resin, is treated with a drop of formalin (which must not contain formic acid) and then with a drop of concentrated nitric acid. The mixture becomes a deep rose-red colour. Avoiding excess of formaldehyde (which causes the production of a greenish mass, and decomposition) the mixture of alkaloid and formalin may be allowed to stand for some hours, when a solid substance is formed which gives a more distinct colour on addition of the acid. Coniine, pyridine, and certain ptomaines which in other respects resemble nicotine, fail to give this reaction.

Cases. In 1902, in New South Wales, an inquest was held on the body of a woman named Doolan in the following circumstances: For some weeks she had complained of abdominal pain and constipation, for which her mistress gave her castor oil with success. Some time later the constipation and pain in the stomach returned, and upon this occasion the unqualified man who was then treating her cut up about an ounce and a half of "Yankee Doodle" tobacco and put it into about a quart of water for a rectal injection. Directly the injection was given the girl went into a "violent fit," foaming at the mouth; the pain in the abdomen became much worse; the bowels acted. She then became very violent, throwing down those who attempted to hold her; a drink of water was then administered but was vomited at once. She died about twenty minutes after the injection was administered. At the autopsy blood was seen issuing from the nose, and the teeth were clenched. There were no signs of disease nor inflammation internally. The stomach and contents, the liver and kidneys, were reserved for examination. An analysis was made by Will. M. Hamlet, Government analyst, who reported that he found no less than six minims of nicotine in the reserved viscera. The verdict was that the woman died from nicotine poisoning, the result of an enema given by G. D. St. Omer, but that the latter was not guilty of wilful negligence or carelessness.

[There seems to be no reasonable doubt that this patient died from nicotine poisoning, but the symptoms were peculiar. She had previously had violent hysterical fits, and it is possible that one of these may have been coincident with the fatal accident, and obscured the symptoms attributable to the tobacco. The fact that so much nicotine was found in the stomach after a rectal injection also requires some explanation.—Ed.]

In the *B.M.J.*, 1895, 2, p. 671, is recorded a curious case of poisoning by nicotine owing to a grape-vine being sprinkled with it. It had probably been actually sprinkled on the grapes themselves.

In the *B.M.J.*, 1901, 1, p. 1544, is an interesting case of tobacco poisoning which arose through a prisoner trying to conceal tobacco in his rectum. He became very ill, but recovered.

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Poisoning by Œnanthe crocata (Water Dropwort)

Source and Method of Occurrence. This umbelliferous plant grows on the banks of rivers, streams, and ditches. It is one of the most poisonous of the order, and is considered to be one of the most virulent of English vegetable poisons. The active principle œnanthotoxin is similar in its action to picrotoxin (*q.v.*).

The root of this plant has large tubers which have been mistaken for edible tubers.

Cases of poisoning by this plant are almost always accidental, but it has been used homicidally.

A case occurred in France in which a woman attempted to poison her husband by mixing slices of the root of this plant with his soup. His suspicions were excited by its acrid taste. The woman was tried for the crime, and Toulmouche deposed at the trial that the plant from which the root had been taken was the *Œnanthe crocata*—that it was a powerful poison, and might cause death in two or three hours. The prisoner was convicted.[1]

Toxicity and Fatal Dose. There can be no doubt that the plant is a powerful poison, but the data are too few to make any dogmatic statement on the quantity required for a fatal result.

Duration. Five minutes from the onset of symptoms seems to be the shortest time on record, and eleven days the longest.

Symptoms. The symptoms are similar to those observed in Picrotoxin poisoning, for example, salivation and vomiting followed by stupor and unconsciousness. Tonic and clonic convulsions then occur with intervals of calm. Death may occur in one of the convulsions from asphyxia. A number of convicts, while engaged at work, ate the leaves and roots of œnanthe. In about twenty minutes one man, without any apparent warning, fell down in strong convulsions, which soon ceased, but left a wild expression on his countenance. Soon afterwards as many as nine of them fell into a state of convulsions and insensibility. The face of the man first seized became bloated and livid, there was bloody foam about the mouth and nostrils, the breathing was stertorous and convulsive, and there was great prostration of strength, with insensibility; he died five minutes after the symptoms had set in. A second died with similar symptoms in a quarter of an hour, although the stomach-pump was used, and some leaves were extracted with the fluids. A third, who had assisted in carrying the two former, was himself seized with convulsions, and died in about an hour; and soon after him a fourth died, in spite of energetic remedial treatment by cold affusion, emetics, stimulants, stimulating frictions, as well as the use of the stomach-pump. Two other cases proved fatal, the one in nine and the other in eleven days; and in these two cases there was irritation of the alimentary canal. In the others who partook of the roots the symptoms were not so severe. Under the free use of purgatives, a considerable quantity of the root was discharged, and a few days later the men recovered.

Treatment. Empty the stomach by repeated washings leaving in a suspensions of animal charcoal. It may be necessary to control the spasms by the inhalation of chloroform. Barbiturates should be administered and the patient protected from all external stimuli. The drug is rapidly destroyed in the tissues.

[1] *Gaz. Méd.*, January 3rd, 1846, p. 18; also *Jour. de Chim. Méd.*, 1845, p. 533.

Post-mortem Appearances. These are not in the least characteristic. On inspecting the bodies of those who died quickly there was congestion of the cerebral vessels ; and, in one instance, a layer of extravasated blood was found beneath the inner membrane (pia mater). In the first case, which proved most quickly fatal, the cerebral vessels were not congested. The pharynx and gullet had a white appearance, and contained some mucus, with portions of the root. The lining membrane of the windpipe and air-tubes was intensely injected with dark blood. The lungs were engorged with fluid blood. The blood in the heart was black and fluid. The stomach and intestines were externally of a pink colour. The cavity of the stomach was lined with a thick viscid mucus, containing portions of the root. The mucous membrane was much corrugated, and the follicles were particularly enlarged. Similar appearances were met with in all.

Analysis. *Œnanthe crocata* can be identified only by its botanical characters. The leaves are of a dark green colour with a reddish coloured border. They have no unpleasant odour when rubbed. The plant bears a greater resemblance to celery than most of the other Umbelliferæ. Its stem is round, channelled, smooth, branched, and grows to the height of two or three feet. The root, consisting of a series of oblong tubercles with long slender fibres, is of a yellowish white colour, and not unpleasant to the taste. It is the most active part of the plant. The leaves yield much tannic acid to water, and potassio-mercuric iodide produces no precipitate in the decoction. The roots and stems of this plant are more frequently eaten than the leaves. Nevertheless it may occasionally be necessary to identify the plant by the leaves.

Case. " J. M., without any previous warning, fell down in a fit in the dining-hall as he was finishing dinner. He regained consciousness soon afterwards. Whilst being removed from the dining-hall to the ward he had a second severe fit, with vomiting. On arriving in the ward his face was livid, his pupils dilated and fixed ; the conjunctivæ did not respond to the touch ; there was a bloody foam about the mouth and nostrils ; the breathing was stertorous, and there was complete insensibility. He had six severe fits subsequently with an interval of a few seconds between them. The convulsion, which was clonic, was general, but attained its greatest intensity in the lower extremities first, next in the upper extremities, and lastly in the facial muscles. He died before a hypodermic of apomorphine had time to act. It was impossible to use the stomach-pump and give emetics by the mouth, owing to the severe and continuous convulsions. Death was due to asphyxia, and the heart continued to beat for a few seconds after respiratory movements had ceased.

" On the same date T.F. was seized with a severe fit and vomited a quantity of food whilst being carried into the ward. He was seen at once, and half an ounce of ipecacuanha wine given, which induced vomiting in a few minutes ; the effects of the emetic were kept up by giving the patient tepid water to drink. There was no insensibility in this case, but there was a marked change in the mental state after the convulsions. The patient was delirious and talked incessantly to himself, was drowsy, and did not like being questioned. His face was pale, the pupils dilated, and the pulse weak and slower than normal. Two hours afterwards he imparted the following information :—

" Between 12·30 and 1 p.m., while at work in a field, he got what he described as a piece of carrot from the patient J. M. He took two bites of this and then threw it into a stream of water at the lower end of the garden. The writer, in company with the head attendant, searched this place and found what looked at first sight like a piece of parsnip in the water. It had a strong disagreeable smell and acrid taste. The broken surface was dotted over with reddish-brown spots each the size of a pin's head. These were not present when another part of the root

was broken across, and only appeared after exposure to the air for a few minutes. The root of one of the plants which were dug up consisted of as many as twenty oblong tubercles, varying in length from four to eight inches. This plant grows in great abundance in marshy places and by the banks of sluggish streams in the south of Ireland, and country people use it for poulticing boils, carbuncles, and other inflammatory swellings. On the following day T. F. complained of pain and a sense of heat in throat, chest, and hypogastrium, also some difficulty in swallowing. On examination the fauces and pharynx were seen to be congested. Castor oil was given, and strong tea after the vomiting had ceased. This was the only medical treatment used in the case." [1]

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Poisoning by Opium (Papaver somniferum)

Source and Method of Occurrence. Opium is the dried juice of the poppy (*Papaver somniferum*) and owes its activity to the presence of a large number of alkaloids. These form two chemically different groups, A, the phenanthrenes to which the narcotic properties of opium are due. These comprise morphine, codeine and thebaine and their derivatives such as dionin, heroin, etc., and B, the isoquinoline group comprising papaverine and narcotine which have no narcotic properties.

Morphine is the chief poisonous alkaloid of opium, of which it forms from 5 to 20 per cent. Good opium should yield 10 per cent. of the alkaloid. The British Pharmacopœia (1932) states that opium in the moist condition as imported must contain not less than 9·5 per cent. of morphine, and dry powdered opium 10 per cent. The official dose of powdered opium is one half to three grains, and of the salts of morphine one-eighth to one-third of a grain.

Morphine, a phenanthrene derivative, possesses two hydroxyl groups, one phenolic and one alcoholic. Replacement of the phenolic by a methoxy group gives *codeine* ; replacement of both hydroxyl groups by methoxy groups gives *thebaine*. Besides these naturally occurring derivatives of morphine, several artificial derivatives have been introduced into medicine with the object of supplying a drug which has the sedative action of morphine without its depressing action on the respiratory centre. These include : *heroine* or *diamorphine* (diacetyl morphine) dose $\frac{1}{24}$ to $\frac{1}{8}$ grain ; *dionine* (ethyl morphine, the ethoxy group replacing the phenolic OH), *dilaudid* (dihydro-morphinone), *peronine* (benzoyl morphine), etc.

Apomorphine which is used as an emetic and has none of the sedative action of morphine, is produced from morphine by the action of moderately concentrated mineral acid.

[1] *B.M.J.*, 1 : 509 (1900).

Various synthetic analogues of morphine (for use as analgesics) have been produced—e.g., metapon, pethidin.[1]

Papaverine is a derivative of benzylisoquinoline and is accompanied by two related alkaloids, *narcotine* and *narceine*. These have little effect on the nervous system (i.e., are neither analgesics nor narcotics) but an antispasmodic action on smooth muscle. In crude opium, it is the effects of the phenanthrene group of alkaloids (and especially morphine) which predominate.

Heroine is probably the favourite narcotic drug for self-medication, and has more addicts, especially in the United States of America, than any other drug. It is free from the constipating effect of morphine.

Pantopon, *Omnopon* and *Holopon* are preparations of all the alkaloids of opium without the other extractives.

Narcophine is morphine-narcotine meconate.

In Taylor's time Winslow's Soothing Syrup, Godfrey's Cordial, Dalby's Carminative, Locock's Pulmonic Wafers and doubtless many other equally pernicious preparations were freely sold by grocers and chemists, and to every one of them many deaths were due, mostly accidental and amongst children. Black Drop and Battley's Sedative Solution, though used by the medical profession, were very dangerous preparations owing to the uncertainty of their strength, which was from two to four times stronger than the official tincture. Chlorodyne and Nepenthe were amongst the most successful remedies of uncertain strength. That state of affairs is now at an end and the sale of all opium preparations is now controlled by means of the Dangerous Drugs Acts (p. 226).

Toxicity and Fatal Dose. The medicinal dose of opium for a healthy adult is from half a grain to three grains. The medicinal dose of the tincture is from five to thirty minims. It contains 1 per cent. of morphine. The fatal dose of opium is impossible to fix owing to its difference in action on different people but in general we may consider that about 30 grains would be fatal. Deaths have been reported from much smaller and recovery from much higher doses. The *smallest dose of solid opium* which has been known to prove fatal to an adult was in the case of a man *æt.* 32, who died very speedily in a convulsive fit after having taken two pills each containing about one grain and a quarter of extract of opium. Dr. Taylor knew of a case in which three-quarters of a grain of opium in solution killed an old woman. The smallest recorded dose of Tr. Opii is two drachms, but even five ounces have been recovered from under treatment. Children are liable to be fatally poisoned by very small doses of the drug.

A child, four months old, was nearly killed by the administration of one grain of Dover's powder, containing only one-tenth of a grain of opium. The child suffered from stupor and other alarming symptoms. In 1822, four grains of Dover's powder, containing two-fifths of a grain of opium, were given to a child four years and a half old. It soon became comatose, and died in seven hours. Thorn met with the case of an infant, only three days old, to which ten grains of Dover's powder, containing one grain of opium, were given by mistake. It suffered from the usual symptoms, but survived for the long period of forty-four hours. Kelso met with an instance in

[1] Small "Chemistry of the Opium Alkaloids". Pub. Health Rep. 1932. Supp. 105.
Small *et. al.* "Studies on Drug Addiction". *ibid.* 1938, Supp. 138.
Eddy, *Amer. J. Med. Sci.*, 1939, *197*, 464.

which a child, nine months old, was killed in nine hours by four drops of laudanum, equal to about *one-fourth of a grain* of opium ; it was much convulsed before death. A case is referred to in the *Medical Gazette* in which two drops of laudanum, equal to about the *seventh of a grain* of opium, killed an infant. A nurse gave to an infant, five days old, *two drops* of laudanum at about three o' clock in the morning. Five hours afterwards the child was found by the medical attendant in a state of narcotism. It was revived by a cold bath, but a relapse came on, and it died the same evening, about eighteen hours after the poison had been given. The fatal dose here, as in the former case, was equal to one-seventh of a grain of opium. An infant, *seven days* old, died in eighteen hours from the effects of *one minim* of the tincture, or *one fifteenth of a grain* of opium.[1] Coma was complete within half an hour. The smallest fatal dose recorded in an infant was in a case communicated by Edwards, of Liverpool (November, 1857). An infant, four weeks old, died with symptoms of poisoning by opium within seven hours after a dose of paregoric elixir, equivalent to *one-nineteenth* of a grain of opium, had been administered to it.

With a knowledge of these facts, it is not surprising that infants are occasionally killed by opium in circumstances in which an adult would not suffer.

In December, 1860, an inquiry took place at Chester respecting the death of a child, aged six weeks, in the following circumstances : A fomentation composed of laudanum and gin was applied to the side of the mother, and the child was put to the breast shortly afterwards. The child fell into a sleep from which it did not awaken, and died, in spite of treatment, the next morning. The cause of death was left obscure ; but it is not improbable that the child drew a quantity of laudanum into its mouth sufficient to cause death.[2]

In some instances infants have been found to manifest an astonishing power of recovery.

An infant of six months recovered after having had administered to it ten grains of Dover's powder, equal to one grain of opium[3] ; and in January, 1852, an infant of nine months recovered under treatment from a dose of two teaspoonfuls of laudanum, given by mistake. This quantity left by evaporation four grains of an impure extract of opium. In 1860, an infant of between two and three months old recovered after *five grains* of opium had been given to it in mistake for rhubarb. Hays met with a case in which a child, not quite six years old, swallowed a powder containing *seven and a half grains of* opium mixed with powdered chalk. The child was not seen until fourteen hours afterwards. It was at first excited. There had been no vomiting. The narcotism was at no time very profound ; it gradually subsided, and at the end of three days the child had recovered.[4]

The Fatal Dose of Morphine. It is as difficult to fix the minimal lethal dose of morphine as it is of opium. The ordinary fatal dose may be placed at about 3 grains but there is great idiosyncrasy to the drug. One grain must be considered highly dangerous although recovery has occurred after a dose of sixty grains. Goodman and Gilman[5] state that 60 mg. of morphine is usually a toxic dose for an adult not in pain, that serious symptoms usually follow the ingestion of 100 mg., and that doses of 250 mg. usually cause death. These figures, of course, apply to non-addicts. Several cases are known in which a dose of *one grain* of hydrochloride of morphine has proved fatal to adults.

Sir Thomas Stevenson met with a case in which two doses, each of half a grain of hydrochloride of morphine, taken internally, are supposed to have killed a hale man in middle life. A case in which *one grain* of the acetate, dispensed by mistake in a pill, caused the death of a lady is

[1] *Lancet*, 1854, 1, p. 419.
[2] *Med. Times and Gaz.*, 1861, 1, p. 70.
[3] *Lancet*, 1850, 18, p. 698.
[4] *Amer. Jour. Med. Sci.*, April, 1859, p. 367.
[5] *Pharmacological Basis of Therapeutics*, 1941, p. 209.

reported. Narcotic symptoms came on within about half an hour, and she died nine hours later.[1] Infants are highly susceptible to morphine.

In no other drug (unless it be cocaine and tobacco) is the **influence of habit** so marked as in opium. Persons may by habit take enormous doses of opium with comparative impunity ; and as much as half a pint of laudanum has been stated to have been taken per diem for long periods. The dose required to kill an *habitué* of the drug cannot even be guessed at, and hence the ordinary fatal dose is not material. Lastly and, from the point of view of the medical profession, most importantly, comes the fact that when we use the drug professionally we are always in a state of uncertainty as to what the result will be, for idiosyncrasy is an important factor ; if severe pain is present (as in renal or biliary colic, etc.) doses can be and are administered with perfect propriety quite outside the official ones, which are useless in these circumstances. In pulmonary diseases, when the bronchi are choked with pus, etc., opium is distinctly contra-indicated owing to the slowness of respiration which it causes, and yet there are times at which the alternative is relief by morphine, or a distressing death. In heart disease, too, morphine is a most valuable drug, and yet it almost seems at times as though it were responsible for death. In fact, in almost any disease, it is impossible to draw a line between doses which are certainly safe and doses which may be lethal.

The **method of administration** has *per se* but little to do with the fatal dose, provided only that absorption can take place. Naturally effects are more rapid and certain when the drug is injected into the blood stream or subcutaneously than when it is ingested.

Morphine is readily absorbed from any mucous membrane and from damaged skin surface but scarcely at all from the intact skin. It is rapidly distributed to the tissues and appears to be readily destroyed by the tissues and particularly by the liver.

Relatively small proportions are excreted into the urine (approximately twenty per cent. partly free, partly conjugated). Gross and Thompson[2] have reported that they have recovered almost all the morphine administered to dogs in the urine, the total execretion is non-tolerant animals being about 90 per cent. and in tolerant animals 65 per cent. Oberst[3] reports similar results in human beings but with only about half the amounts found in dogs. A small quantity, 5 per cent., is excreted in the faeces. A more negligible quantity appears to be excreted into the stomach although it was formerly believed that excretion into the stomach was of serious dimensions.

Small quantities of morphine may continue to be excreted for twenty-four hours and possibly longer.

Duration. Since opium has no local irritant action, the symptoms only occur after some of the poison has been absorbed. Hence it is easy to understand the great variation in the time of onset.

The symptoms usually *commence* in from *half an hour* to *an hour* after the poison has been swallowed. Sometimes they come on in a few minutes, especially in children, and at other times their appearance is postponed for a long period. When morphine is hypodermically injected,

[1] *Pharm. Jour.*, July, 1872, p. 16.
[2] *Journ. Pharm. & Exp. Therap.*, 72 : 138 (1941).
[3] *Journ. Pharm. & Exp. Therap.*, 73 : 401 (1941).

symptoms appear within three or four minutes. In a case reported by Skae, the person was found totally insensible in *fifteen minutes*. As we might expect from the facts connected with the absorption of poisons, when the drug is taken in the *solid* state, the symptoms are commonly more slow in appearing than when it is *dissolved* in water or alcohol.

In once case observed by Sir Thomas Stevenson a dose of twenty grains of morphine was fatal in about half an hour, but most cases of poisoning by opium or morphine prove fatal in from about six to twelve hours. Those persons who recover from the stupor, and survive longer than this period, generally do well ; but there may be a partial recovery, or a remission of the symptoms, and afterwards a fatal relapse.

In 1843, a gentleman swallowed a quantity of laudanum, and was found labouring under the usual symptoms. The greater part of the poison was removed from the stomach by the pump, and he so far recovered from his insensibility as to be able to enter into conversation with his medical attendants ; but a relapse took place, and he died the following night. Such cases are not uncommon, and danger cannot be considered to be absent for at least twenty-four hours.

The Hon. Mrs. Anson (January, 1859) swallowed, while fasting, an ounce and a half of laudanum by mistake. In a quarter of an hour emetics were given, but she did not vomit for half an hour ; and she was not treated medically for two hours and a half. The matter then drawn from the stomach had no smell of laudanum. She was quite unconscious, and had lost the power of swallowing. After remaining in this comatose state for upwards of nine hours, the patient revived. The face became natural, the pulse steady. The power of swallowing returned. She was able to recognise her daughters, and, in a thick voice, to give an account of the mistake she had made. This state lasted about five minutes ; the torpor then returned. She again sank into profound coma, and died in fourteen hours after the poison had been taken.

The symptoms, however, generally progress steadily to a fatal termination ; or the stupor disappears, vomiting ensues, and the person recovers. Several instances are recorded of this poison having destroyed life in from seven to nine hours. One occurred within the author's knowledge in which an adult died five hours after taking the drug. Christison met with a case which could not have lasted above five, and another is mentioned by him which lasted only three hours. Procter communicated to the author the case of a female, *æt.* 50, who swallowed an ounce of the pharmacopœial tincture, and died from the effects in less than *two hours*. Opium was found in the stomach. It is possible that the drug may destroy life even with greater rapidity than this ; but, as a medico-legal fact, we are at present only entitled to state that it has destroyed life within the short period above mentioned. On the other hand, the cases are sometimes much prolonged. There are several instances of death in fifteen or seventeen hours and Hunt [1] reports a case in which a woman took about 7 grains of morphine and $\frac{1}{6}$ grain atropine. She was unconscious when found at 7.30 a.m. and died the next morning at 10.45 a.m. The time between the ingestion of the poison and death was placed at approximately thirty-three hours.

Symptoms. The symptoms which manifest themselves when a large dose of opium, or any of its preparations, has been taken, are in general of a uniform character, but now and then anomalous symptoms occur, due no doubt to the different alkaloids present in raw opium ; for example, thebaine has not only a narcotic, but also a convulsant action, and its narcotic action is masked by its effect on the cord. Narcotine also has a

[1] *B.M.J.*, June 14, 1913.

much greater action on the cord than morphine has. Papaverine exerts an antispasmodic effect on plain muscle but has little, if any, narcotic effect. However, the typical symptoms of opium poisoning are identical with those of morphine poisoning.

A feeling of well being and ease is early felt. There may be a certain pleasurable mental excitement in some people usually of very short duration. The period of excitement is followed by weariness, headache, incapacity for exertion, a sense of weight in the limbs, diminution of sensibility, giddiness, drowsiness, a strong tendency to sleep, and stupor succeeded by perfect insensibility, the person lying motionless, with the eyes closed as if in a sound sleep. In this stage he may be roused by a loud noise, and made to answer a question ; but he speedily relapses into stupor. In a later stage, when coma has supervened with stertorous breathing, it will be difficult, if not impossible, to rouse him. The pulse is at first small, quick, and irregular, the respiration hurried, the skin warm and bathed in perspiration, sometimes livid ; but when the person becomes comatose, the breathing is slow and stertorous, and the pulse slow and full. As the coma deepens the respirations become slower and may show the Cheyne-Stokes type. The pupils in the early stage are strongly contracted ; in the last stage, and when progressing to a fatal termination, they may be found dilated. They are commonly insensitive to light. The expression of the countenance is placid ; the eyes are heavy, the lips are livid and cyanosis gradually becomes more pronounced. Sometimes there is vomiting, or even purging ; and if vomiting takes place freely before stupor sets in, there is great hope of recovery. This symptom is chiefly observed when a large dose of opium has been taken ; and it may then, perhaps, be ascribed to the mechanical effect of the poison on the stomach. The odour of opium is occasionally perceptible in the breath. Nausea and vomiting, headache, loss of appetite, and lassitude, often follow on recovery. In cases likely to prove fatal, the muscles of the limbs feel flabby and are relaxed, the lower jaw drops, the pulse is feeble and imperceptible, the sphincters are relaxed, the pupils are unaffected by light, the temperature of the body is low, there is a loud mucous rattle in breathing, and convulsions are sometimes observed before death, but more commonly in children than in adults. One of the marked effects of this poison is to suspend all the secretions, except that of the skin. Even during the lethargic state, the skin, although cold, is often copiously bathed in perspiration. The **contracted state of the pupils** has been considered to furnish a valuable distinctive sign of poisoning by opium or the salts of morphine. In relying upon it, it is necessary to bear in mind the fact that in hæmorrhage into or pressure on the pons varolii the pupils are also contracted. In carbolic acid poisoning the pupils are much contracted, though seldom so minutely as in opium poisoning ; and there is coma and stertorous breathing. As a rule the peculiar odour of carbolic acid in the breath will prevent any mistake as to the nature of such a case. The condition of the pulse varies greatly. It has been found small and feeble, sometimes full and slow. In some cases there is great irritability, as well as itching of the skin and irritability of the bladder, with difficulty of passing urine. In one case of morphine poisoning, an apothecary was able to walk for an hour and a half after taking seventy-five grains of hydrochloride of morphine.[1]

[1] Hays, *Amer. Jour.*, October, 1862.

It has been stated that opium in large doses may sometimes operate not as a narcotic, but as a stimulant to the nervous centres, causing violent convulsions. In some instances the convulsions are said to have assumed a tetanic character, resembling those caused by strychnine. This is stated to have been noticed where the alkaloid or its salts have been used hypodermically. It has been alleged that the tetanic symptoms of strychnine are not to be distinguished from those caused by large doses of morphine. One medical authority announced that all the symptoms assigned to poisoning by strychnine in Cook's case[1] might be explained by supposing that he had taken three grains of morphine. [Considering that upwards of fifteen alkaloids and analogous bodies have been extracted from opium and that the actions of some of these are distinctly convulsant, there is no difficulty in accepting the above statement as representing an exceptional case in which the victim was peculiarly sensible to the convulsant bodies in opium, so that their action overcame that of the morphine. Such a possibility need not offer any forensic difficulty now that the extractives of opium are better understood.]

Treatment. If the drug has been taken by the mouth, the stomach should be well washed out by means of the stomach-tube, using warm water in which permanganate of potash has been dissolved in sufficient quantity to colour it pink. If animal charcoal is available a tablespoonful should be stirred into the water used for lavage. In default an emetic may be administered by the mouth, or apomorphine may be injected subcutaneously. Emetics, however, usually fail owing to the depression of the vomiting centre. If morphine has been administered hypodermically there is only a trace of excretant into the stomach and repeated gastric lavage may lead to a serious chloride depletion.[2] Persevering attempts are to be made to rouse the patient by external stimulation; the faradaic current, applied to various parts of the body with a wire brush, is an efficacious stimulant: walking the patient to and fro between two assistants may be resorted to, but only in the less severe forms of poisoning. When the coma is profound, artificial respiration must be adopted and persisted in as long as the heart continues to beat; this constitutes a most *valuable aid to recovery*, for it must be remembered that death is due to failure of the respiratory centre. A better method of stimulating respiration is to supply air containing about 5 per cent. of carbon dioxide. It may be supplemented by faradaic stimulation of the phrenics. Ammonia may be applied to the nostrils in the form of smelling-salts. Hot coffee may be given by the mouth if the patient can swallow; if not, it may be administered by the stomach-pump or as an enema. The patient must be kept warm for the drug tends to lower the temperature of the body. One-fortieth of a grain of atropine sulphate, injected hypodermically and repeated if necessary, has been recommended for the purpose of stimulating the respiratory centres, but its utility is doubtful, notwithstanding the number of successful cases in which it has been used (see section on "Antagonism of Poisons"). The injection of caffeine is safer and more useful. Lucatello had a case in which a patient swallowed about forty-five grains of opium and twenty-two grains of morphine sulphate on an empty stomach. Symptoms did not appear

[1] *R. v. Palmer.*
[2] Goodman and Gillman, *Pharmacol Basis of Therepeutics* 1941, p. 211.

for an hour. Breathing having nearly ceased, artificial respiration, and faradaisation of the phrenics, were resorted to, but without effect; under the influence of hypodermic injections of strychnine, respiration was resumed.

Coramin (5 c.c.) given slowly intravenously has proved effective in the hands of many observers. It may be repeated at intervals of thirty minutes.

In cases which are not very severe, the cold douche and perambulating the patient may be sufficient, but the former should never be used when the surface is cold, nor should the latter be carried to excess so as to exhaust the strength. *In severe cases it is worse than useless to drag a comatose individual about.*

Post-mortem Appearances. There is nothing characteristic unless it be the smell of opium in the stomach contents. With morphine this is of course not present. In a case which proved fatal in fifteen hours, the vessels of the head were found unusually congested throughout. On the surface of the fore part of the left hemisphere of the brain there was an ecchymosis, apparently produced by the effusion of a few drops of blood, and there were numerous bloody points on its cut surface. Fluidity and a dark colour of the blood are mentioned as common appearances in cases of poisoning by opium. There is often also engorgement of the lungs. Among the external appearances there is often great lividity of the skin. The stomach is so seldom found otherwise than in a healthy state that the inflammatory redness said to have been occasionally seen may have been due to accidental causes. When tincture of opium has been taken and retained in the stomach, increased redness of the mucous membrane may be produced by the alcohol alone.

Analysis. *Opium.* There are no means of detecting opium itself, either in its solid or liquid state, except by its smell and other physical properties. The smell is said to be peculiar, but a similar smell is possessed by lactucarium, which contains neither meconic acid nor morphine. The odour is a good concomitant test of the presence of the drug, whether it be in a free state or dissolved in alcohol or water, but it is not perceptible when the solution is very much diluted, and it loses its characteristic smell by exposure. The odour passes off when an opiate liquid is heated; it also escapes slowly at room temperatures. Again, it may be concealed by other odours, or the drug may undergo some change in the stomach during life that may rapidly destroy the odour. The analysis in cases of poisoning by opium is therefore limited to the detection of morphine and other alkaloids and the meconic acid with which they may be combined; and in this respect it must be remembered that meconic acid is found *only* in opium or narcophine which is derived from opium.

Colour Test for Indian Opium. Porphyroxine, described by Merck in 1837 as the red colouring matter of opium, is according to Hesse a mixture of several bases, one of which is meconidine, and another probably rhœadine, which latter alkaloid also occurs in the capsules and other parts of the red poppy. Kanny Lall Dey[1] believes porphyroxine to be a chemical individual, and Dakshit[2] describes it as carbonyldihydrocodeine.

[1] *Pharm. Jour.*, 1882, 3, 12, 397.
[2] *Ber.*, 1926, 59B, 2473.

Kanny Lall Dey[1] states that by treating the aqueous extract of Indian opium with ammonia or sodium carbonate, and immediately agitating with ether, the ethereal solution always leaves on evaporation a substance which, when warmed with dilute hydrochloric acid, gives a rich purple coloration, and he recommends the reaction as a test for Indian opium. Turkey and Smyrna opium do not give the reaction. Merck repeatedly dips a slip of filter paper in the ethereal solution, allowing it to dry spontaneously after each immersion. The paper is then moistened with hydrochloric acid and exposed to steam, when it will acquire, especially after drying, a more or less distinct rose-red colour.[2] With Turkey and Smyrna opium no such reaction is obtained.

Morphine. Morphine has the following properties :—(1) It crystallises in fine white prisms. These crystals may be obtained by adding weak ammonia to a solution of morphine in hydrochloric acid. (2) It is only very slightly soluble in cold water, of which 3,330 c.c. dissolve 1 gram of alkaloid at 25° C., and is not very soluble even in boiling water (1 gram in 460 c.c.). The aqueous solution has an alkaline reaction. The salts of morphine, however, are usually soluble in water. Morphine is very slightly soluble in ether, thus differing from narcotine ; but it is dissolved by fifty parts of cold alcohol, and about thirty parts of boiling alcohol. It is dissolved by a solution of potash or soda, in which narcotine is insoluble, and from this solution morphine cannot be completely removed by ether. It is soluble in ethyl acetate and in amyl-alcohol, and these have been employed as substitutes for ether in extracting morphine from organic liquids. (3) It is easily dissolved by a very small quantity of all dilute acids, mineral and vegetable. (4) Morphine and its solutions have a bitter taste. (5) The salts of morphine are not precipitated in a crystalline form by solutions of sulphocyanide of potassium, ferrocyanide of potassium, or of chromate of potassium. In this respect they are strikingly distinguished from the salts of strychnine, which give well-marked crystalline precipitates with these three reagents.

Tests for Morphine. Since a number of putrefactive bases are known to give reactions similar to those of certain alkaloids—of which morphine is one—it must be emphasised that reliance must never be placed upon a single test, and that *identity* of behaviour of the suspected material and a known sample of the alkaloid should be a *sine qua non*. Reference may be made to the cases of Urbino de Freitas in Portugal, of Songzogna in Italy, and of Buchanan in America, in all of which controversy arose as to confusion between morphine and putrefactive bases.

(1) *Marquis' Test.* The reagent is 3 c.c. pure concentrated sulphuric acid to which two drops of 40 per cent. formaldehyde solution have been added. The dry alkaloidal residue on a white porcelain dish is touched from the point of a glass rod with this reagent, when a fine purple-red colour, changing to violet and then to blue, is produced. A marked colour is given by 0·02 mgrm. of the alkaloid.

Codeine and apomorphine give violet and then blue, but not the initial purple. Dionine gives a dark blue violet. Heroine gives the same colours as morphine, and dilaudid behaves very similarly.

[1] *Pharm. Jour.*, 1882, 3, 12, 397.
[2] Allen's "Commercial Analysis," vol. 6, p. 403.

(2) *Deniges' Test* (modified by Oliver). To one or two drops of a slightly acid solution of a morphine derivative (morphine, heroine or dilauded) add a drop of hydrogen peroxide and make alkaline with ammonia. Stir with a bright piece of copper wire. A brisk effervescence ensues, and a wine-red colour develops. This may be masked by a blue colour due to the copper, and in such cases potassium cyanide should be added to discharge the blue colour and reveal the characteristic red. Codeine and dionine give no colour.

(3) *Apomorphine Reaction* (*Husemann's Test*). A fragment of the residue is warmed on a water bath with a few drops of concentrated sulphuric acid, in a watch-glass, for about half an hour, thus converting morphine to apomorphine. A brownish or almost black residue appears. Cool, and add one drop of concentrated nitric acid. Tilt the watch-glass so as to spread the nitric acid over the whole area of residue. A red-violet or blue colour is produced, changing rapidly to dark blood red and then to yellowish red.

A preferable modification is to moisten the residue with pure concentrated sulphuric acid, allowing it to stand for twenty-four hours in a desiccator, then add a drop of concentrated nitric acid, when the red violet colour appears, changing to dark blood red and then to yellow red. A crystal of potassium nitrate or chlorate may be substituted for the nitric acid. Codeine and heroine give the same colours.

(4) *Nitric Acid.* Add one or two drops of the strong acid to a fragment of the solid. The morphine is entirely dissolved, and the solution acquires a deep orange-red colour. Warm this solution till the red colour turns yellow, dilute with a little warm water and add gradually a fresh solution of sodium thiosulphate ; the colour is destroyed, and does *not* change to violet (distinction from brucine). Heroine gives a yellow colour on addition of nitric acid, and, on warming, this changes to bluish-green, then almost blue, and finally yellow. This test is stated to be characteristic of heroine. Codeine gives a yellow colour which does not turn red.

Oliver states that codeine, narcotine, heroine and dionine fail to give this reaction, but Webster finds that both heroine and dionine may give deep red colours.[1]

(5) *Iodic Acid with Chloroform.* A drop of solution of iodic acid is mixed with twice its volume of chloroform. There should be no change of colour. On adding a small quantity of this mixed liquid to morphine or its salts, either solid or in solution, the iodine is separated from the iodic acid and dissolved by the chloroform, which sinks to the bottom, acquiring a pink or red colour, varying in its intensity according to the quantity of morphine present. The presence of morphine may be thus easily detected in one drop of the tincture of opium, in chlorodyne or other opiate liquids, in spite of the presence of organic matter. If chloroform is not used, iodine may be detected by the blue colour produced on the addition of a solution of starch. The colour is discharged by solution of ammonia, and the supernatant aqueous liquid acquires a brown colour. No reliance can be placed on the liberation of iodine alone. This test is of value only as a negative test, though Lefort claims the mahogany-brown colour to be

[1] Peterson, Haines, and Webster, "Legal Medicine and Toxicology," 1923, Vol. II p. 534.

characteristic of morphine. It may, however, easily be masked by pre-existing colour of an impure preparation. Heroine and codeine do not liberate iodine from iodic acid.

(6) *Sulphomolybdic Acid.* Dissolve, with a gentle heat, 1 or 2 mgrms. of powdered molybdic acid in 1 c.c. of *pure* concentrated sulphuric acid, and cool. The liquid should be freshly prepared, and kept from contact with air and organic matter. When one or two drops are rubbed with dry morphine or any of its salts an intense reddish-purple or crimson colour is produced. This changes to a dingy green and ultimately to a splendid sapp' ire blue. A minute trace of morphine is thus revealed. Heroine behaves somewhat similarly, dilaudid gives a pale mauve which soon fades, and codeine, dionine and narceine give bluish colours which in the case of codeine develops from an initial yellow. This test produces no rapid change in *strychnine*, but the mixture slowly acquires a pale blue tint. The presence of morphine in strychnine is thus easil; detected. When poured on *brucine* the solution becomes a rose-red, changing to greenish brown and ultimately dark blue. When mixed with *veratrine* the liquid becomes greenish brown, and gradually passes to a darker shade. The margin becomes purple, and ultimately the whole mixture acquires a deep blue colour.

(7) *Iron Test.* A drop of a neutral solution of ferric chloride added to a fragment of morphine produces a blue colour (greenish with excess of reagent), destroyed by HCl or by heat. Heroine and codeine do not give the test; dila :did gives a blue, and apomorphine reddish brown.

(8) Solid morphine is mixed with two to eight times its weight of powdered cane sugar (or a mixed solution of the two substances is evaporated to dryness) and a drop of concentrated sulphuric acid is added. A purple colour is produced, and changes gradually to blood red and brownish red, becoming olive brown on dilution with water. The colour is not extracted by chloroform. Codeine behaves similarly.

Meconic Acid. This is a solid, colourless, crystalline acid. It is combined with morphine in opium, of which it forms on an average 8 to 10 per cent.; and it serves to render this alkaloid soluble in water and other menstrua. *Tests.* There is only one test upon which any reliance can be placed, namely, *ferric chloride* or *ferric sulphate*. This test produces, even in a dilute solution of meconic acid, a deep blood-red colour; and it is owing to the presence of this acid that a salt of iron gives a red colour with tincture and infusion of opium as well as with all liquids containing traces of meconate of morphine, the effects of the iron test with morphine being counteracted by the presence of meconic acid. The red colour of ferric meconate is not easily destroyed by boiling nor by dilute mineral acids (distinction from ferric acetate), nor by a solution of corrosive sublimate (distinction from ferric sulpho-cyanide), but it is by sulphurous acid and stannous chloride. The colour, destroyed by stannous chloride, is restored by addition of nitrous acid (sodium nitrite and dilute hydrochloric acid). In liquids containing tannic acid, *e.g.*, tea or beer, the action of this test is obscured by the production of ferric tannate. The dark colour is removed by a few drops of dilute sulphuric acid. Meconic acid is partly extracted from acid solution by ether, and can thereby be separated from tannins, etc.

Detection of Opium in Organic Mixtures. Opium itself may be regarded as an organic solid, containing the poisonous salt which we wish to extract. It is not always in fatal cases of poisoning with opium or its tincture, even when these are taken in large quantity and death is speedy, that we can succeed in detecting meconate of morphine in the stomach. It is probably removed by vomiting and by absorption. If the matter is solid, it should be cut into small slices; if liquid, evaporated to a syrup; and, in either case, digested with a large quantity of rectified spirit, slightly acidified with acetic acid. The residue should be well pressed in linen; the alcoholic liquid should then be evaporated at a low temperature until it is dry. The residue should be digested in absolute alcohol, filtered, and the filtrate evaporated to dryness as before. The residue is to be taken up with water, filtered, and treated with acetate of lead until there is no further precipitation. This liquid should be warmed and filtered; lead meconate is left on the filter, while any morphine passes through. The surplus acetate of lead contained in the filtered liquid (containing the morphine) should now be precipitated by a current of sulphuretted hydrogen, the sulphide of lead separated by filtration, and the liquid evaporated at a very gentle heat, so that any sulphuretted hydrogen may be entirely expelled. On treating this extract with alcohol the acetate of morphine, if present, will be dissolved. The alcoholic liquid is again evaporated and taken up by water. On evaporating a drop or two of the water liquid with a drop of freshly prepared solution of starch, and touching the residue with a drop of a solution of iodic acid, morphine will reveal itself by the production of a blue colour. Other tests must then be applied to the solution or to the residue on evaporation.

The lead *meconate* left on the filter is readily decomposed by warming it with a small quantity of dilute sulphuric acid; and in the filtered liquid, neutralised if necessary by an alkali, the meconic acid is detected by the ferric chloride test. A current of sulphuretted hydrogen may be used in place of sulphuric acid. The sulphide of lead takes down with it much of the organic matter of the precipitate. This analysis requires care as well as some practice in the operator, in order that the morphine should be obtained in a sufficiently pure state for the application of the tests.

Before resorting to this process it is advisable to employ *trial tests* on the original liquid, in order to determine whether any meconic acid or morphine is present or not. The smell of opium may be entirely absent. Meconic acid may be readily detected by the action of a ferric salt on the diluted organic liquid, and morphine may be found by adding to a portion of the liquid a mixture of iodic acid and chloroform. The chloroform acquires a pink colour by dissolving the iodine set free by morphine or its salts. These tests may equally be applied to a solution of opium obtained by dialysis.

Chronic Poisoning by Opium – Drug Addiction

Opium and its alkaloids and derivatives, notably morphine and heroine, are frequently used for their narcotic effect alone. This addiction showed signs of becoming more common after the 1914-18 war, but the Dangerous Drugs Acts and regulations have made it more difficult to obtain these drugs and have caused a diminution in the number of addicts. It would appear that heroine is much more liable to form a habit than morphine, and when the habit is formed, it is harder to break with heroine.

The most prominent predisposing cause of addiction appears to be inherent mental or nervous instability. Other causes are chronic pain, insomnia and various states of insanity. It is occasionally taken from curiosity or from imitation, but habits thus acquired are not common, and are relatively easily cured.

Opium may be taken in many forms, smoked, ingested or injected. The opium smoker is uncommon in Great Britain, and as the amount of the drug absorbed is very small, opium smoking rarely produces the disastrous effects seen in those who inject the drug. Opium eating and drinking is also much more common in the East than amongst Europeans.

In Europe and America the drug is usually taken hypodermically, either morphine or heroine being the alkaloid chosen. Heroine is more popular than morphine, probably because it has less constipating effect. When the habit is once formed, the drug has to be continuously increased to secure the narcotic action. Tolerance is soon acquired, and fifteen to twenty grains per day is by no means a large dose in a confirmed morphine addict.

Symptoms. The use of opium or its alkaloids is first followed by a feeling of well-being, but as the habit grows and larger doses are taken, this feeling of well-being is soon lost and depression follows. In addition there is a constant dread of the crisis of withdrawal. The patient becomes irritable, and sleep is disturbed by terrifying dreams, or there may be insomnia. Loss of memory and mental fatigue are common. There is a gradual deterioration of the highest centres of the brain, and a gradual change in character and conduct. Lying, stealing and other evidences of moral deterioration are thus observed. Hallucinations may occur. The face is haggard, the colour bad, the pupils usually contracted. Gastric disturbance, constipation, loss of appetite, emaciation and weakness, headaches and dizziness are frequently observed. The physical condition in heroine addiction is, as a rule, less prominent than when morphine is used. When the supply is constant and the drug has not been pushed, the addict may present quite a normal appearance.

Treatment. Treatment to be successful should be carried out in an institution.

Withdrawal of the drug is the first step. This may be abrupt in the case of opium eaters and smokers, and in most patients of robust constitution. Such a procedure is impossible in the chronic hypodermic addict, especially if he is nervous and emaciated.

In these cases it must be withdrawn gradually by reduced doses so that zero is reached in about ten days.

It is essential to make certain before withdrawal commences that the patient gives up the whole of his supplies. He should also be placed under careful supervision to prevent him from securing fresh supplies, which he will certainly do when the drug is withdrawn if it is possible.

Free purgation, treatment of the gastric acidity, nutritious and easily digested food, graduated exercises, baths and massage are required. The insomnia must be relieved by the use of veronal, sulphonal, paraldehyde and other hypnotics, using different drugs at different times. The use of hyoscine has been strongly recommended. Relapses are common.

Withdrawal Symptoms. If the habit is not of long standing the withdrawal symptoms are mild, and consist of uneasiness, depression, weakness and restlessness. In older cases there may be very severe symptoms if the drug is completely withheld. There is nausea, yawning, sneezing and coryza, dilatation of the pupils, violent pains and cramps in the abdomen and legs. There may be irritability and excitably which may progress to a condition of mania. There may be severe collapse with weak and irregular pulse, and death may occur from cardiac failure. Injection of morphine causes an immediate arrest of the symptoms.

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Poisoning by Physostigma venenosum

Source and Method of Occurrence. The calabar bean is a large leguminous seed of dark colour, resembling a garden bean, but much thicker and more rounded in its form. It is brought from the western coast of Africa, where it is employed by the natives as an ordeal bean for the trial of persons suspected of witchcraft.

The common belief is, that the innocent vomit and are safe, while the guilty retain the poison and die from its effects. So strong is popular confidence in this test that those who are suspected voluntarily take an emulsion of this seed, and, as Christison remarks, many an innocent person thus pays the penalty of his rash reliance on this superstitious custom. It is a firm belief that if a man dies he is guilty.

The seed or bean has a thin, hard, dark-coloured, brittle covering ; the kernel inside is white, and weighs from thirty-six to fifty grains, the whole seed about sixty-seven grains. This bean owes its properties to the presence of an alkaloidal substance called *physostigmine* ($C_{15}H_{21}N_3O_2$) or *eserine*, which is found in the cotyledons, associated with several other alkaloids of similar nature. The seeds contain about

0·25 per cent. of alkaloid. Christison found that the active principle could be extracted by alcohol, which dissolves 2·7 per cent. of the seed, including this substance. The greater part of the seed, as in nux vomica, consists of starchy and other inert matters, with a small quantity of oil. The kernel is yellowish white, without bitterness, acrimony, aroma, or any distinct impression on the organ of taste. In fact, it cannot be distinguished by taste from a haricot bean.

The drug is now largely used in ophthalmic practice in the form of lamellæ, each containing $\frac{1}{1000}$ grain of physostigmine salicylate.

It is also official in the form of Physostigmine Salicylate, of which the dose is 2 mg., orally for an average adult, or 0·5 to 1·0 mg. hypodermically. It has been used in the treatment of gastro-intestinal atony (*e.g.* paralytic ileus), and myasthenia gravis, though a synthetic analogue, *prostigmine*, is often preferred in these conditions.

Toxicity and Fatal Dose. A drop of a solution of physostigmine applied to the eye produces within from ten minutes to a quarter of an hour a remarkable contraction of the pupil. This has been observed to last in children for fifteen or twenty hours. In this respect the poison is eminently distinguished from the natural mydriatic alkaloids, atropine, hyoscyamine, and cocaine, which cause dilatation of the pupil. It causes contraction of the pupil when taken internally, as well as when applied locally (*vide* also case below).

The special character of poisoning by calabar bean consists mainly in a diminution of the excitability, and paralysis of the peripheral nerves, and paralysis of the excito-motor centres of the heart and their peripheral endings, also in producing contraction of the vessels and of the unstriped muscles, especially those of the intestine and the sphincter muscles of the iris, as well as in promoting salivary and other secretions. It has but little, if any, action on the brain cortex, as is evidenced by the retention of consciousness until failing cardiac and respiratory activity produces coma and death.

Six beans have proved fatal. Recovery has taken place after intravenous injection of a quarter of a grain of eserine sulphate (*see* case).

Symptoms. Amongst the first effects observed are weakness in voluntary movements restlessness and muscular twitching. The secretions, perspiration, saliva, tears, etc., are increased. Vomiting is common. There is dyspnœa, the pulse is slow, the blood pressure raised, and the pupil contracted. [In certain reported cases of poisoning, however, the pupils have been found dilated.] Consciousness is retained for some time, and death appears to be due to asphyxia. Desiring to try the effects of this seed on himself, Christison took the eighth part of a seed, or six grains, one night before going to bed. There was a slight sense of numbness in the limbs during the night, but in the morning no urgent symptoms of any kind. He then chewed and swallowed the fourth part of a seed (twelve grains). Twenty minutes later he was seized with giddiness, and a general feeling of torpor over the whole frame. He immediately swallowed an emetic, and thus emptied his stomach. The giddiness, weakness, and faintness increased to such a degree that he was obliged to lie down in bed. In this state he was seen by two medical friends, who found him prostrate and pale, the heart and pulse extremely feeble and tumultuously irregular, the mental faculties intact, extreme faintness threatening dissolution,

but no apprehension of death on the part of the patient. There was no uneasy feeling of any kind, no pains nor numbness, no prickling, not even any sense of suffering from the great feebleness of the heart's action. There was the will but not the power to vomit; the limbs became chill, with a vague feeling of discomfort. Stimulants were employed, and warmth and pulsation, with a power of moving gradually returned. Two hours after the poison had been taken he felt drowsy, and slept for two hours more, but with such activity of mind that he had no consciousness of having been asleep. The tumultuous action of the heart continued. After this the symptoms gradually disappeared, and the next day he was quite well.[1]

In 1864, fifty children were poisoned at Liverpool by eating a quantity of these beans. The sweepings of a ship from the west coast of Africa had been thrown on a heap of rubbish; the children found the beans and ate them. A boy, *æt.* 6, who ate six beans died within a very short time. The principal symptoms were severe griping pains, constant vomiting, and contracted pupils. In addition to these symptoms, the face was pale, and the eyes were bright and protruding. In attempting to walk, the children staggered about as if they were drunk. In 1864, two children, aged six and three years respectively, chewed and ate the broken fragments of the kernel of one nut. Within about forty minutes they complained of sickness. One child held his head drooping, appeared sleepy, and his hands were powerless. He staggered, and was scarcely able to walk. He complained of severe pain in the stomach, and made ineffectual attempts to vomit. Milk was given, and he then vomited. The child became quite prostrated; the pulse was feeble and slow; and the pupils were slightly contracted. Some pieces of the nut were thrown up by the vomiting. The other child had pain in the abdomen, and was listless, sleepy, and depressed. He vomited freely, some portions of the nut being ejected. He could neither stand nor walk. His face was pale, the eyes were piercing, but the pupils and pulse were natural. In this case there was purging. The children recovered on the third day.[2] In cases in which it has proved fatal to animals it has caused much irritation and congestion of the stomach and bowels (Dragendorff).

African travellers have described the symptoms resulting from the use of the beans as an ordeal as consisting of violent thirst, inability to swallow, cramps, and muscular twitchings. If vomiting takes place, as it often does early when a large dose is given, recovery ensues rapidly, otherwise consciousness remains till shortly before death, which occurs usually within half an hour.

Treatment consists of gastric lavage with weak permanganate solution and giving one to two mg. of atropine sulphate subcutaneously or intravenously (according to the urgency). This usually counteracts the dangerous effects of physostigmine on the circulatory and respiratory systems, though not the muscular twitchings which are harmless and gradually disappear. Artificial respiration may be necessary.

Post-mortem Appearances. There are none peculiar to the plant unless it be the extreme contraction of the pupils. Judged from the

[1] *Pharm. Jour.*, 1855, p. 474.
[2] *Edin. Med. Jour.*, 1864, p. 193.

case below, this might be absent, and then there only remains discovery of portions of the bean in stomach or vomit and the chemical analysis of the viscera.

Eserine is rapidly eliminated by the saliva and other secretions.[1]

Analysis. Physostigmine (eserine) combines with acids to form salts. It is slightly soluble in water, easily so in alcohol, ether and chloroform.

Bromine Test. A solution of bromine in water acts in a characteristic manner on a solution of the sulphate, producing a red colour when $\frac{1}{1000}$th of a grain or even less is present (the colour of the bromine itself interferes to some extent).

Ammonia Test. When a fragment of the alkaloid or one of its salts is evaporated to dryness on a water bath with excess of ammonia solution, it turns successively red, yellowish red, yellow, green and blue, finally leaving a blue or blue-green residue. The residue dissolves in ethyl alcohol with a blue colour which dyes silk without a mordant, and stains the skin. Excess of dilute mineral or acetic acid added to this solution changes the colour to red, and the solution shows a strong reddish fluorescence. Examined spectroscopically, the blue alkaline solution shows one absorption band in the red, while the red acid solution exhibits one absorption band in the yellow. On treating the blue solution with reducing agents, such as hypophosphorous acid, hydrogen sulphide, sulphurous acid, etc., the solution first becomes red, and is then decolorised.

Rubreserin Test. Aqueous solutions of eserine become red and then dark brown, on exposure to air and light. This is due to the formation of a red crystallisable colouring matter called rubreserin, which is soluble in chloroform. Reducing agents decolorise this reddened solution. If the colourless aqueous solution is shaken for some time with excess of sodium or potassium or ammonium hydroxide, it rapidly acquires a pink-red colour. On agitating this with chloroform, the red colouring matter rubreserin is dissolved out and colours the chloroform orange red.

Phosphomolybdic acid gives with solutions of this alkaloid the usual yellowish precipitate, and this dissolves in ammonia to a deep blue solution as in the case of morphine. A drop or two of fuming nitric acid added to a boiling solution of the alkaloid gives a gamboge colour, which changes to greenish blue on evaporation or intensely violet on the addition of caustic alkali. The violet colour is changed to wine red by dilution with water or to pale orange by acid (whence it is restored by alkali).

The *physiological test* consists in the application of a solution of the alkalold to the eye. It produces strong contraction of the pupil when this liquid contains but a minute proportion of physostigmine.

It may be extracted from organic material by the method described on p. 265 for alkaloids.

Cases. The following case is reported by Leibholz :—

"Two girls, aged twenty-four and eighteen respectively, obtained possession of a sealed tube containing 0·1 gramme of physostigmine sulphate, which they dissolved in water, and each girl drank half of the solution. For half an hour they pursued their household avocations without experiencing any effects ; they then suddenly became unconscious. In each case the face was red and shining ; the pupils, dilated to the maximum, were reactionless ; the pulse, sixty to the minute,

[1] Husemann's *Jahresber.*, 1872, p. 570.

was full and of high tension ; the respirations were shallow, rapid, and moaning : pain was experienced in the region of the stomach and abdomen ; vomiting occurred early, and persisted for some time after return to consciousness. Dilatation of the pupils, with feeble reaction to light, lasted for several days, perfect recovery ultimately taking place. The activity of the alkaloid was vouched for by Merck after chemically examining a companion sample. Tested physiologically, three milligrammes injected under the skin of a rabbit weighing four pounds produced paralysis of the voluntary muscles, difficulty of respiration, violent diarrhœa, and death in ten minutes. A solution dropped into the human eye caused marked contraction of the pupil. The dilatation of the pupils in the above-recorded cases is remarkable. Cases of calabar bean poisoning have occurred without contraction of the pupils, but none with dilatation. The absence of diarrhœa is in marked contrast to its universal occurrence in animals poisoned with physostigmine."

The following case[1] reported by Slater is of interest :—

M. S., aged thirty-two, was admitted to the London Hospital suffering from psoriasis. Inadvertently the patient was given a quarter of a grain of eserine sulphate in fifteen minims of water by intravenous injection. Within a minute she became unconscious, with laboured breathing, imperceptible pulse, and cyanosis, fibrillary twitchings of all muscles, and wide dilatation of the pupils. There was incontinence of urine and fæces, and a great flow of saliva, and the throat and larynx were filled with secretion. Immediately the mistake was recognised an injection of atropine $\frac{1}{100}$ grain and strychnine $\frac{1}{60}$ grain was given, and oxygen was administered by inhalation. An hour later the pulse could just be felt, the throat was clearer, but breathing was still laboured. A second injection of atropine $\frac{1}{100}$ grain was given, and the inhalation of oxygen continued.

Shortly afterwards the patient began to regain consciousness, but it was at least an hour later before she spoke. She complained of deafness and blindness. The breathing was now calm and deep, and the twitchings had almost stopped. The pulse was weak and irregular. The oxygen was stopped ; saline (two pints) was injected by the rectum and retained. Gradually the sight and hearing returned, and the general condition steadily improved. The following day the patient felt rather weak, but was otherwise none the worse.

The course of the psoriasis appeared not to be affected by the incident.

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Poisoning by Pinus (sp. var.), or Oil of Turpentine (Oleum terebinthinæ)

Source and Method of Occurrence. The oil is distilled from the oleoresin, the natural exudation of the trees. Oil of turpentine has caused a few fatal cases of poisoning among children. It is used as a cutaneous counter irritant (in liniments) and as an expectorant. As an athelmintic it is obsolete.

Toxicity and Fatal Dose. Turpentine cannot be said to be very toxic. Goodman and Gilman (Pharmacological Basis of Therepeutics, 1941, p. 820) give 15 cc. for children and 150 cc. for adults as possibly fatal doses. It may irritate the stomach as a primary effect, and after absorption it has a specific influence on the kidneys, causing irritation which may lead to inflammation. Maund records the death of a woman within a few hours of taking about 6 ounces of turpentine.[2]

[1] *B.M.J.*, December 9th, 1922.

[2] *Lond. Journ. Med. Sci.*, 1858, 36, 561 (quoted by Peterson, Haines and Webster "Legal Medicine and Toxicology," 1923, Vol. II., p. 696).

It is to be noted, however, that the most dangerous doses of turpentine are the ones intermediate between the official two to ten minims and the anthelmintic dose of half an ounce. The former can be dealt with by the kidneys, and the latter is generally carried off by the purgation produced, but doses of one or two drachms are insufficient to purge, and may act very deleteriously upon the kidneys; indeed, a case of death from doses of one to two drachms was some years ago the subject of a judicial inquiry, in which the medical man was severely blamed.

Symptoms. The principal symptoms are nausea, vomiting, colic and diarrhoea from its irritant effect on the gastro-intestinal canal; delirium, excitement and ataxia from its effect on the central nervous system after absorption; pain in the lumbar region and pain on micturition with blood and albumen in the urine from its irritant effect during excretion. In a dose of three drachms it has produced intoxication. A child was given a tablespoonful by mistake; within three hours there was complete insensibility, with stertorous breathing, strongly contracted pupils, rapid and weak pulse, coldness of the surface, paleness of the countenance, general relaxation of the muscles, and occasional convulsive movements. Several fatal cases are recorded. One was the case of a child aged fourteen weeks. The child had had half an ounce of the oil poured down its throat by a brother, *æt.* 8. It had been left asleep at 9 p.m., and within an hour it was found to be insensible, cold and slightly convulsed. At midnight it was comatose, pale, with extremely cold surface, contracted pupils; slow and irregular breathing, about three times in a minute; pulse quick, small, compressible, almost imperceptible. A strong odour of turpentine issued from the mouth, and there was a spot of liquid on the pillow. The child was unable to swallow. It died within fifteen hours after taking the poison. In another case the child was five months old. A spoonful of spirit of turpentine was given to it in mistake for peppermint, and death took place rapidly.[1]

Tests. The odour of the drug is characteristic. If a few drops of the oil are stirred in a porcelain dish with a drop of hydrochloric acid and a drop of ferric chloride and warmed, a pink colour changing to violet red and then to blue is produced. If the oil is allowed to stand with concentrated hydrochloric acid, terebenthene dihydrochloride (insoluble in water) crystallises in rhombic plates.

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Poisoning by Primula obconica (Chinese Primrose)

Source and Method of Occurrence. This plant, introduced from China, is frequently grown in gardens and greenhouses. Many years ago attention was drawn to the danger of handling it, and numerous cases of very severe illness from it have since been reported. The

[1] *Pharm. Jour.*, July, 1872, p. 75.

following, reported by Dr. Leighton, is a fairly typical though severe case (*B.M.J.*, 1898, 2, 1159) :—

" In October, 1896, I was called to G. W., aged forty, head gardener on an estate near. I found him suffering from tremendous œdema of the right hand, with well-marked lymphangitis of right arm, and tenderness in the axillary glands. Next day there began a profuse serous discharge from several points on the back of the hand, and from between the second and third fingers. The pain in the joints was excessive, and the patient was slightly feverish. The discharge continued for eight days, gradually decreasing.

" In June, 1897, the patient had a similar attack, all the symptoms of the first being reproduced, only with less severity. This attack yielded to treatment in a week.

" In September, 1898, a third attack came on, this time in the right foot—pain, œdema, and slight serous discharge from dorsum of foot. The discharge was much less than in the two attacks in the hand, and on the fourth day the swelling had completely disappeared.

" *Diagnosis.* When called in the first time I could discover no skin abrasion whatever, and inquiry failed to trace any of the usual causes of local blood poisoning. Careful questioning, however, disclosed the fact that a few days previously the patient had handled a number of plants of the *Primula obconica*. These plants were subsequently banished from the greenhouses. In the second and third attacks there was again no abrasion on hand or foot, and the patient had handled none of these plants since October, 1896. I concluded, therefore, that the poison must still be in the tissues in spite of the lapse of time (two years) from its first effect.

" *Treatment.* In each attack I applied a carbolic poultice locally, and administered liquor ferri perchlor. internally. The symptoms began to abate from the commencement of treatment in each case, having up to then gradually got worse.

" *Note.* The time between the first and second attacks was eight months, between the second and third sixteen months, indicating a gradual elimination of the absorbed poison, also proved by the lesser severity of the symptoms."

Another case is reported in the same volume, p. 1472, and other cases in the *Lancet*, 1896, 2, p. 1802, *et alia*.

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Poisoning by Ranunculus (sp. var.)

Source and Method of Occurrence. The buttercups form one of the glories of our English flora, and considering the acrid and irritant properties they possess, it is rather remarkable that so few cases of poisoning by them are recorded. Although popularly supposed to be freely eaten by cows, it is more probable that cows as a rule avoid them, as may easily be seen in any meadow where *R. acris* grows freely. The white water buttercups are said to be innocuous, and are eaten freely by cattle when opportunity offers.

One fatal case has been recorded,[1] but no details are given except that an inquest was held on a boy who died with all the symptoms of irritant poisoning a few hours after eating some buttercups.

[1] *Lancet*, 1897, 1, p. 1781.

Poisoning by Rhus toxicodendron and other Species of Rhus

Contact with certain species of Rhus cause severe toxic skin reactions. The active principal in all species is an oily resin toxicodendrol which exudes in the sap when any part of the plant is damaged. It does not appear to be volatile and actual contact is required. *Rhus toxicodendron* L. (poison ivy). *R. venenata* D.C. (poison sumach) and *R. diversiloba* (poison oak). are the common plants but there are many others. The symptoms appear after an incubation period of four to five days as a rule although it may be as little as one or as long as seven days. Symptoms consist of erythema. hyperæmic macules. papules. vesicles and pustules. secondary lesions being excoriations and ulcers. Subjective symptoms are extreme itching and a burning stinging heat. There may be some fever and symptoms of kidney irritation. In a large number of cases the chief parts involved are the back of the hands, insides of forearms, eyes, ears and genitalia.

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Poisoning by Ivy (Hedera helix)

The following case of poisoning by ivy is recorded by Turton[1] :—

A boy, aged three and a half years, was, according to his mother, taken ill on June 20th at midday. I saw him two and a half hours later. He was then in a condition of mild delirium, which alternated with stupor. He never completely lost consciousness, and could be roused when he relapsed. During the delirious stage clonic convulsions would come on, and he screamed and cried. No tetanic spasms were observed. He could not stand. He had visionary hallucinations that bulls were chasing him and rats and beetles crawling over him ; they lasted for many hours. An intense scarlatiniform rash, most marked on the legs, face, and back, was present. There was no vomiting or purging. The pulse was rapid, but full and bounding, becoming weaker later. The pupils were widely dilated. The temperature was raised.

Emetics were given, but failed to act, and the stomach was washed out. Afterwards he was given a stiff dose of castor oil, which was retained, but did not act until some hours later.

The symptoms abated after the wash-out, and in about three hours he was fairly well. When the bowels acted the cause was discovered—namely, ivy leaves. He had eaten a considerable quantity of these. Next morning he confessed to having eaten the ivy leaves, and a boy who was with him corroborated. The ivy was the common climbing variety.

A point of interest was the close resemblance which the symptoms showed to belladonna poisoning—namely, the irritation of the central nervous system followed

[1] *B.M.J.*, August 15th, 1925.

by depression, the widely dilated pupils, and the scarlet rash. The distinguishing point was the absence of dryness of the mouth and throat which invariably follows poisoning by belladona. In view of the fact that *Rhus toxicodendron* may cause a skin rash it is interesting to note the presence of a rash in the case.

Poisoning by Ricinus communis (Castor Oil Plant)

Source and Method of Occurrence. The oil is obtained from the seeds of a plant named *Ricinus communis*.

These seeds contain an extremely active toxic protein, *ricin*, which however does not pass into the expressed oil.

Toxicity and Fatal Dose. The smallest exactly recorded fatal dose of the seeds is two.[1] The oil itself can hardly be said to have any toxic properties, for its ordinary dose is about an ounce for an adult, with a rather disproportionately large allowance for children, as it is a most suitable purgative for them. The cake left after the pressing of the oil is poisonous to rats as well as to human beings. It contains ricin in comparatively large amount. When the seeds are swallowed whole they may fail to produce the severe symptoms described below.

Ricin, the toxic principle in the ricinus seeds, was isolated by Stillmark in 1889, and was regarded by him as a toxalbumen. This is probably correct, for although exceedingly poisonous solutions have been obtained (*e.g.* by Jacoby) which gave no protein reactions this may be due to the extremely small amounts required to produce poisoning. A great deal of experimental work on ricin has been done since Ehrlich's discovery (1891) that this substance, like the true bacterial toxins, is antigenic, *i.e.*, its injection into the body calls forth the production of a highly specific antitoxin (antiricin). The toxicity of ricin is very great. Ehrlich used solutions of which 1 : 200,000 was the minimal lethal dose for the mouse. One-thousandth part of a milligramme will frequently kill a guinea-pig.

In general it may be considered that about five of the seeds are fatal for children and about twenty in adults, but much smaller doses cause severe toxic symptoms.

Duration. In the recorded fatal cases death has followed only after some days' interval. In the fatal case from two seeds (above) the patient, a dock labourer, succumbed on the sixth day. However susceptible an animal may be, there is always an incubation period before the outbreak of symptoms and this may amount to several days.

Symptoms. At the time of eating the seeds there is an absence of any disagreeable taste or sense of heat in the mouth and throat but after a varying period there is severe pain in the abdomen, copious and painful vomiting, with bloody purging, thirst, and convulsions, terminated by death. A girl, *æt.* 18, ate about twenty, one of her sisters four or five, and another two castor oil seeds. About five hours after the seeds were eaten the girl felt faint and sick ; vomiting and purging came on and continued throughout the night. On the following morning she appeared like one affected with malignant cholera. The skin was cold and dark coloured, the features contracted, the breath cold, the pulse small and wiry ; there was restlessness, thirst, pain in the abdomen, and she lay in a sort of drowsy, half-conscious state. Whatever liquid was taken was immediately

[1] *Pharm. Jour.*, 1900, p. 305.

rejected, and the matters passed by stool consisted chiefly of a serous fluid with blood. She died in five days without rallying; the two other sisters recovered.

An officer took seventeen seeds as a purgative. Three hours later there was violent purging, followed by vomiting and severe cramps, the patient passing into a condition resembling the collapse of Asiatic cholera. The vomiting was not stopped until after twenty-one hours and recovery then took place. There was suppression of urine for forty-eight hours.

Two children, aged respectively six and three years, ate some castor oil seeds. The children when brought to the hospital were suffering from extreme collapse, consequent on vomiting and purging, the bodies pale and perspiring, pulse 130. The stools were frequent and watery. The substances ejected from the stomach were pulpy; there was pain in the abdomen, great thirst, and the tongue was furred and dry. There were no cerebral symptoms. Under treatment, they both recovered in two days.[1]

In experiments, at the end of at least twenty-four hours the animal appears quiet and almost somnolent. Later, convulsions, diarrhœa and coma supervene with general failure of the respiration and death.

Treatment must be on general principles (*vide* pp. 25) *et seq.*).

Post-mortem Appearances. The bowel is inflamed and there may be many ecchymoses. Blood may be found in the serous cavities and extravasations in the internal organs. Patches of necrosis may be found in the liver and in the intestinal wall.

In experimental animals constant lesions are found in the alimentary canal and consist of extreme congestion with swelling of the lymphoid nodules, ecchymoses and thrombosis of the vessels, especially in the mesentery. The viscera are congested, and there is a widespread parenchymatous degeneration in the liver, kidney and myocardium.

Analysis. Castor Oil *seeds* can be identified in the contents of the stomach provided a portion of their outer coat is obtained. These seeds are remarkable for their peculiarly variegated surface. Externally they are pale grey, marbled with yellowish-brown spots and stripes.

In animals killed by injections of ricin, the ricin is apparently eliminated by the bowel, as the liquid contents in its lumen, rendered sterile by filtration, are capable of setting up ricin intoxication in a healthy animal (Stepanoff). A remarkable action of ricin demonstrated by Stillmark is exerted on suspensions of blood corpuscles in saline solution. An agglutination of the red blood corpuscles rapidly takes place, and they sink in the form of dense flocculi to the bottom of the test-tube, which affords a biological test for the presence of the poison.

The ricin (in stomach contents or saline extract of seeds) may be partly purified by dialysis or ultra-filtration, and the final preparation, with salt content adjusted to that of physiological saline and *p*H adjusted to 5·6 or 9·0 is added to a saline suspension of red blood cells in a small test-tube or microscope slide,[2] other toxalbumins behave similarly. For identification by the specific precipitation tests, see Bamford. "Poisons," 2nd Edition, 1947, Chapter XII.

[1] *Med. Times and Gaz.*, 1870, 1, 581.

[2] Inoue. J. Soc. Chem. Ind. Japan, 1937, 40, 12; cf Analyst, 1937, 62, 567

Cases. The following case, though happily not fatal, corroborates the fact that two seeds are sufficient to give rise to severe symptoms. The case is reported by Dr. Burroughs :—

" Shortly after two o'clock on August 19th, a porter, aged about fifty, employed on the London and North-Western Railway at Euston station, was brought to my house in a cart, obviously very ill. He then told me that while sweeping out a railway truck he had picked up and eaten two seeds which he said he took to be ' kernels ' of some kind. Half an hour after he had eaten them he was overcome with giddiness to such an extent that he was unable to stand, and shortly after vomited eight times in rapid succession.

" Some half-hour later, when he was brought to me, he complained of throbbing in the temples, pains in the head, giddiness, nausea, slight fulness and pains in the stomach, and a sense of dryness and constriction in the throat. His face was very pale, his forehead and cheeks covered with beads of sweat ; his lips were blue, his pupils dilated, and his whole face bore a pinched and anxious expression. The rest of the skin was moist and cold. His respiration was slightly quickened, and pulse 84.

" He now told me that he had two ' kernels ' similar to those which he had eaten in his possession, which I thereupon examined and took to be rather dried-up specimens of the seeds of the castor-oil plant, both from their external appearance and, on breaking the outer husk, from the peculiarly unpleasant odour suggestive of the well-known flavour of the oil. No sooner had he handed these to me than he again vomited, and in the matter thus expelled I found several portions of the beans. I then hoped that, as he had already vomited profusely before, this might prove to be the last of the poisonous material ; but as his condition did not improve, I evacuated the contents of the stomach, discovering two more small fragments mingled with some blood-stained mucus, after which he seemed greatly relieved. As no further symptoms of poisoning supervened, and his condition steadily improved, I sent him home and saw him on the following day, when he said that he felt comparatively well, but rather weak.

" Dr. Stevenson confirmed the fact that the seeds were those of *Ricinus communis*."[1]

For another fatal case from two seeds, *vide B.M.J.*, 1900, 1, p. 317. The following case occurred in 1904 [2] :—

A.L., *æt.* 25, the subject of gleet, on his way to work at 3 p.m. on October 24th, 1904, saw some castor-oil beans. Being unaware of their nature and properties, he picked up and ate about twelve. At first they seemed to taste " rather like walnuts," but after he had eaten this number a somewhat " sickly taste " developed.

About 5 *p.m.* (*two hours later*), he was taken ill with violent abdominal pains ; obstinate and very painful vomiting and retching every five minutes ; vomit " like green treacle," no blood ; frequency of micturition (no hæmaturia), every five minutes, five to eight ounces ; violent purging for one hour every five minutes ; stools " like green treacle," no melæna.

About 7.30 *p.m.* (*four and a half hours later*) violent cramps in his legs, which began to draw up, and felt " as if they had lumps in the calves."

About 8 *p.m.* (*five hours later*) : Dizziness, began " to lose himself " ; seen by a doctor and sent to hospital.

Condition on admission (*about five and a half to six hours later*) : Face pinched, lips blue, temperature normal, pulse small, low tension, cold extremities, much collapse ; on striking abdomen, muscles went into violent tonic contraction ; calves contracted violently when cramps came on.

Treatment. Washed out at once ; castor oil ℥ss. statim ; calomel grs. iv. statim ; R pot. brom. grs. xx. ; chloral grs. x. ; aq. ad ℥ss. ; ℥ss. *per rectum* 4tis horis ; hot bottles to extremities ; hot fomentations to abdomen ; nutrient enemata.

Course : Obstinately constipated after entering hospital ; pains increased till twelve hours after eating beans ; gradually subsided.

Three days later : Retching still persisted every fifteen to twenty minutes, pains much less severe ; pulse still rather rapid and small, though much improved.

Five days later : Pains and retching stopped ; pulse fairly good.

[1] *B.M.J.*, 1903, 2, p. 836.
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Poisoning by Rumex (sp. var.)

Source and Method of Occurrence. *Rumex acetosa* and *acetosella*, with which must be placed *Oxalis acetosella*, are common weeds not infrequently eaten as salad. Fatal poisoning by them is a most rare event. One of the few cases to be traced is reported by Dr. Suckling in the *Lancet* of July 31st, 1886. Professor Eichhorst, of Zürich, at a clinical meeting reported a case of a boy, aged twelve years, who was taken ill on April 26th and who died on May 5th, 1899. The illness commenced with gastro-intestinal symptoms and hæmorrhagic nephritis, and was diagnosed on the first day. The patient eventually succumbed to uræmia. On the day before being taken ill the boy had eaten large quantities of common sorrel (*Rumex acetosa*), and the most careful questioning of the relatives and the attending physician did not bring to light any other etiological factor. Professor Eichhorst mentioned that there were no cases to be found in German literature, but that French veterinary surgeons report fatal cases in horses due to over-consumption of this herb. In these circumstances it seems particularly important that the public should be made aware of these facts, as common sorrel is considered innocuous and happens to be a favourite dish of boys and girls in early summer.[1] The three plants mentioned all contain traces of oxalic acid, to which their noxious effects are attributed.

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[1] *Lancet*, 2, 1899, p. 60.

Poisoning by Sium latifolium and S. angustifolium or S. nodiflorum : Water Parsnip

Source and Method of Occurrence. This plant is not unlike watercress, for which it has been eaten by mistake. In 1882, two girls, aged five and three years respectively, died from eating the leaves. The day after these were eaten the younger child became suddenly ill and died, while the other succumbed two days afterwards.[1]

Poisoning by Solanum (sp. var.)

Source and Method of Occurrence. There are various species of this plant, including the *Solanum dulcamara*, *Bitter-sweet*, or *Woody Nightshade*, which has a purple flower and bears red berries ; and the *Solanum nigrum*, or *Garden Nightshade*, with a white flower and black berries.

In common with other members of the natural order *Solanaceæ* these plants may contain alkaloids of the atropine group and certain symptoms which arise may be attributed to these alkaloids. It is more likely, however, that the toxic effects will be produced by the alkaloid solanine which is allied to saponium. Such symptoms include pain in the abdomen, vomiting and diarrhœa. The symptoms are often associated with those of atropine and the pupil may be dilated and the pulse rapid and the mouth dry.

The berries of the *Solanum nigrum* produced serious effects in three children who had eaten them, and who complained of headache, giddiness, sickness, colic, and tenesmus. There was copious vomiting of a greenish-coloured matter, with thirst, dilated pupils, stertorous breathing, convulsions, and tetanic stiffness of the limbs. One child died in the acute stage ; the others died apparently from secondary consequences during treatment.[2] From three to four berries of this plant have been found to produce sleep. The berries of the *Woody Nightshade* are stated to have caused the death of a boy, *æt.* 4, in the following circumstances. He had eaten some of the berries, and at first did not appear to suffer from them ; but eleven hours afterwards he was attacked with vomiting, purging, and convulsions, which continued throughout the day, the child being insensible in the intervals. He died convulsed in about twenty-four hours. The vomited matters were of a dark greenish colour and of a bilious character. Other children had partaken of the berries at the same time, but among these only one suffered slightly.[3] In 1892, a death was recorded from eating these berries, which, however, were not clearly identified.[4]

Toxicity and Fatal Dose. Death in all these cases was supposed to be due to the presence of solanine, which is found in the plants. It is a compound (glycosidal) alkaloid which has not yet been thoroughly investigated, but is undoubtedly a poisonous body, and is probably associated with atropine in the Solanum group.

[1] *B.M.J.*, 1882, 2, p. 26.
[2] Orfila, *op. cit.*, 4th ed., 2, 273.
[3] *Lancet*, 1856, 1, p. 715.
[4] *Pharm. Jour.*, August 13th, 1892, p. 139.

Potato Poisoning (Solanum tuberosum)

The potato is too well known as a domestic vegetable to need any description, but it is not so well known that this edible tuber is the product of a plant in all parts of which a very poisonous alkaloid exists. The plant is a member of the *Solanaceæ* family, and the alkaloid solanine is to be found in the flowers, stems, seed, and even in the " peel " of the potato itself. In general, the amount of solanine in potatoes is very small, but it increases if old potatoes are allowed to germinate, and fairly high concentrations may be reached in green potatoes (0·06 per cent.).

The ordinary symptoms of solanine poisoning are headache, severe nausea, vomiting and cramps in the muscles. A fatal issue is rare.

Cases of poisoning occur from time to time and a particularly full account is given by Willimott[1] of an outbreak of potato poisoning in Cyprus.

F. Wallis Stoddart, Public Analyst, Bristol, reported the following case to Sir Thomas Stevenson, on August 27th, 1902 :—

" Two children in succession died in one house after a short illness. The first was *in extremis* when seen, and, as the *post-mortem* revealed nothing, was decided by the medical attendant to have died of ' ptomaine poisoning.' However, when the second case occurred suspicion arose, and an analysis was ordered. The symptoms were very indefinite—some vomiting, history of one or two convulsions, general sort of collapse, and failure of the heart. I was told ' one pupil was slightly dilated,' whatever that means. The viscera were very carefully removed, and the stomach and intestines opened by me in presence of the doctor. There was a little diffuse reddening of the lesser curvature of the stomach, and an injected condition of the lower large bowel, which last, I think, was caused by the tube of a syringe used to wash out the bowel during life. There was no solid matter whatever, but the whole mucous surface was coated with what I found to be a sort of emulsion of castor oil, a dose of which had been given. I received, however, also some undigested matters washed out of the rectum before death, and described to me as gooseberry skins. There were some of these, also pips, etc., but most of the pieces were potato skin and thin flat slices of the starchy tissue such as is removed in peeling. There was evidence also of some germination in one of the ' eyes.' I could find absolutely no poison except a little of our old friend copper in the liver, and gave a very guarded opinion that death was probably due to solanine poisoning, due to eating raw potato peel ; but I feel very far from confident about it."

On the occurrence of solanine in the potato as described in Kunkel's " Vergiftung " Mr. Stoddart remarks :—

" The large increase in solanine in ' bad ' potatoes is odd. The fact that the alkaloid occurs in quantity in the tissue immediately under the skin is of special interest to my case, as this part, the white portion of the ' peel,' was present in large proportion to other matters. The only point of discrepancy I note is that dilatation of the pupils seems a common symptom in poisoning by potato, though doubtfully characteristic of solanine. In the Bristol case the doctor reported that ' one pupil was slightly dilated,' but that is not very definite to my mind."

On this case the *Lancet*, 1902, 2, p. 693, thus comments :—

" It is undoubtedly the case that there is a time when the potato may contain an important quantity of poison, and this appears to be when the tuber has begun to germinate and to shrivel. In that case the solanine has been found chiefly in the peel and at the root of the eyes or shoots. An interesting account of the symptoms produced by eating diseased potatoes appeared in the *Lancet* as far back as 1846 (February 14th, p. 190). A peculiar affection was invariably traced to the use of diseased potatoes, being ushered in by rigors, hot skin, quick pulse,

[1] *Analyst*, 1933, *58*, 431.

and abdominal pain. In the next stage rose-coloured patches appeared and as suddenly vanished, and in the majority of cases diarrhœa supervened: in the third stage there was a swollen state of the muscles of the neck, shoulders, and arms, with pain so acute that the patient winced on the slightest pressure. Inability to raise the arms, pains in all the bones, a red erysipelatous state of the face and skin, with œdema of the eyelids so as nearly to close them, were also observed. Ten cases of this affection had occurred in three or four days in the same locality (in Ireland), and all were similarly affected. The case recorded recently need give no grounds for alarm, for an actively poisonous potato is quite a rare specimen. The poison, however, probably occurs normally in the tuber in very small quantity, but this quantity may increase to a poisonous amount in the sprouting, shrivelled, or diseased potato, and then most of it is found in the skin and eyes. In any case diseased potatoes would be naturally avoided, and they are generally discoloured. The risk, of course, would be greater when such potato is eaten with, as it is often cooked in, its jacket.

"The methods of determining solanine in potatoes have been improved by Professor Schmiedeberg and Dr. Meyer in Strasburg, but no case of poisoning by potatoes has hitherto been described where a quantity of solanine sufficient to produce decided symptoms has been found on chemical analysis. Professor Pfuhl, chief of the hygienic laboratory of the Army Medical Academy of Berlin, has recently published in the *Deutsche medicinishe Wochenschrift* a series of cases occurring in a regiment of the garrison of this city. Between May 29th and June 1st fifty-six men of this regiment were reported ill, the symptoms being those of acute gastro-enteritis. The disease began with a rise of temperature to 38° or 39·5° C. (100·4° or 103° F.), headache, colic, diarrhœa, and general debility. In some cases there was vomiting, in others nausea only; several fainted, and one man was seized with convulsions. The majority were drowsy and apathetic; on the following day their conjunctivæ were yellow, and in one case there was general jaundice. A number of them complained of a feeling of tickling in the throat, the mucous membrane of which was slightly swollen. Other complications were herpes labialis and salivation. There was no mydriasis. The fever continued till the third day. There were from four to six motions of the bowels each day, the fæces being diarrhœal in character and containing no potatoes, but pieces of undigested plums which had formed part of the men's dinner. In two cases the fever reappeared after two days, but after one or two days the temperature became normal again. The men were kept in bed and were treated with abdominal wet packs, three-decigramme (four and a half grains) doses of calomel, and afterwards with laudanum. Nearly all recovered in a few days. It was found that on May 29th, being Whit-Sunday, a portion of their dinner had consisted of plums and potatoes, the potatoes having been quite recently supplied to the kitchen of the company. The plums proved to be normal, and the potatoes were therefore carefully examined. They were large white, round ones, comparatively few of which had sprouted. They had been boiled for twenty-five minutes. On chemical analysis of the potatoes solanine was found to the extent of 0·38 part per 1,000 in the boiled and 0·24 part per 1,000 in the raw. On an average every man who fell ill had 0·3 gramme (four and a half grains) of solanine, a quantity sufficient to produce toxic effects. The rest of the potatoes were, of course, not used; and accordingly no other cases of poisoning occurred." [1]

In the Glasgow outbreak in 1917 (sixty-one cases, one fatal), Harris and Cockburn (*Analyst*, 1918, 43, 133) found 0·041 per cent. of solanine in the potatoes, and during the 1922 epidemics in Germany, amounts varying from 0·01 per cent. to as much as 0·79 per cent. were reported. In one outbreak, on the other hand (*Münch. med. Woch.*, 1903, p. 2282), the symptoms were shown to be due to *B. proteus vulgaris*.

Tests. Solanine is nearly insoluble in water, and insoluble in ether or chloroform but soluble in amyl alcohol. With Mandelin's reagent it gives a yellow colour which changes through orange to purple red, brown, red, violet and blue green, finally fading altogether. Concentrated sulphuric acid gives an orange colour, changed on warming to violet and brown. Marquis' reagent gives a bluish-violet which turns brown very quickly and then slowly changes to an olive-green.

[1] *Lancet*, 1899, 2, p. 1554.

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Poisoning by Strychnos nux Vomica

Source and Method of Occurrence. The plant *Strychnos nux vomica*, from which the seeds are obtained and imported, is not a native of this country. The seeds are flat rounded discs about the size of a shilling, covered with radiating silky fibres, and slightly depressed in the centre. They are of a light brown colour, very hard, tough, and difficult to pulverise. The powder is of a grey-brown colour, like that of liquorice or jalap. It is sometimes met with in a coarsely rasped state. It owes its poisonous properties to the presence of the alkaloid *strychnine*, to the extent of about 1 per cent., associated with another alkaloid named *brucine*, of similar but feebler poisonous properties, in a somewhat higher percentage. The average yield of the two alkaloids is about 3 per cent. Ignatius beans (*S. Ignatii*), another common source, contain about 2 per cent. of strychnine and half that amount of brucine. Strychnine has a very bitter taste even in very small quantity; but as it destroys life in a small dose, and it may be given in the form of pills, or professedly administered as quinine or other medicine, it offers every facility for criminal administration.

The pharmacopœial preparations of nux vomica and strychnine are: Of nux vomica itself, nux vomica powder (1·2 per cent. strychnine), liquid extract (1·5 per cent. of strychnine), dose one to three minims, a dry extract (5 per cent. strychnine), dose one-quarter to one grain, and the tincture (0·125 per cent. of strychnine), dose ten to thirty minims; of the alkaloid the only preparation is the liquor strych. hydrochlor. (one grain in 110 minims), dose three to twelve minims and Syrup Ferri Phosph. cum Quinine et Strychnine, " Easton's Syrup " (0·025 per cent. strychnine). The official dose of strychnine hydrochloride is $\frac{1}{32}$–$\frac{1}{8}$ grain.

Strychnine may be accidentally mixed with ordinary medicaments. In 1891 two adults died at intervals of several weeks after the purchase in London of Epsom salts at a particular shop; and in each case it was ascertained that death was due to strychnine. In 1888, a gardener murdered his wife and son by means of pills containing strychnine, substituted for ordinary purgative pills.[1]

Sir Thomas Stevenson knew extract of nux vomica to be sold and taken in mistake for extract of sarsaparilla with fatal result. No strychnine may now be sold in rat poisons or in any other way to the public, except as an ingredient in a medicine.

A certain number of deaths take place from the accidental ingestion of various preparations of strychnine such as tablets of Easton's Syrup, etc.[2]

In April 1904, at Barrow, a child found a box of pills made up to contain the dose of a drachm of Easton's Syrup. Of these he ate ten, and soon died in convulsions. This is a very typical case of (*a*) the dangers of disguising nauseous drugs by modern pharmacy, and (*b*) the carelessness with which potent drugs are left about.

[1] *R. v. Bowles*, C. C. C., January, 1888.
[2] Aikman, *J. Amer. Med. Assoc.*, 1930, *95*, 1661. Ross and Brown, *Canad. Pub. Health J.*, 1935, 26, 237.

Toxicity and Fatal Dose. The medicinal dose of strychnine for an adult ranges from $\frac{1}{32}$–$\frac{1}{8}$ of a grain. One-thirtieth of a grain has caused the death of a child between two and three years of age in four hours. Three-quarters of a grain killed a child, *æt.* $7\frac{1}{2}$, in half an hour.[1] In two cases of adults, in each of which a quarter of a grain had been taken by mistake, the patients recovered only under early treatment. The smallest fatal dose in an adult was in the case of Dr. Warner. *Half a grain* of the sulphate of strychnine here destroyed life.[2] So powerful are the effects of this drug in certain cases, that ordinary medicinal doses can scarcely be borne. A gentleman took one-twentieth of a grain of strychnine in each of six doses during a period of two or three days. Severe fits of tetanus occurred, although half a grain had not been taken altogether. It is probable in such cases that elimination is either arrested or imperfectly performed. Tweedie prescribed pills for a gentleman each containing one-fifteenth of a grain of strychnine. He took altogether five of them, or one-third of a grain, at intervals. The patient was seized with alarming tetanic convulsions, continuing for some time. There was also opisthotonos of a severe kind. He slowly recovered. In two cases Sir Thomas Stevenson has found doses of *one-twelfth* and *one-fifteenth* of a grain of strychnine to produce tetanic convulsions. A *fatal dose* of strychnine for an adult may be assigned at from **half a grain to two grains,** although it has been stated that in America deaths have resulted from one-quarter of a grain.

Harvey Littlejohn[3] described the following cases of strychnine poisoning, which are of considerable interest :—

A naval officer of twenty-five wanted a "pick-me-up" and was given one drachm of liq. strychninæ hydrochloridi by a ship's doctor who misread the dose printed in his Pharmacopœia. After twenty minutes the officer laughingly complained, "That was strong stuff. I can't walk properly now." The doctor became suspicious, discovered his error, and gave the officer salt and water and tartar emetic, which produced some vomiting. In spite of this, and of artificial respiration, the officer died from respiratory failure three-quarters of an hour after taking the poison and after only one convulsion. One drachm of liq. strychninæ hydrochlorici contained 0·55 gr. of strychnine.

A boy, aged three and a half, was found playing with some tabloids of Easton's Syrup. After an interval he had convulsive seizures and died on arrival at hospital not more than one and a half hours after taking the tabloids. As far as could be ascertained, he had taken two or possibly three tabloids, equal to a total of one-thirty-second or one-twentieth of a grain of strychnine.

Mr. John Webster quoted a case of tolerance to the drug in which a woman took increasing doses of strychnine for two years before her death. In the last few weeks these doses rose from one-tenth to two-fifths of a grain per day without the appearance of symptoms, perhaps partly owing to the presence of paraldehyde in the prescription. *Post-mortem* analysis showed only very faint traces of strychnine in the liver and stomach, too small to estimate, although the large doses were taken up to death.

As in other cases of poisoning, many recoveries have taken place after much larger doses. There are several instances on record in which persons have recovered after taking one grain or more. A case of recovery from two or three grains is reported.[4] A girl recovered in six or seven hours from a dose of *four grains* of strychnine.[5] When first seen, she

[1] *Ann. d'Hyg.*, 1861, 1. 133.
[2] "On Poisoning by Strychnia," pp. 138, 139.
[3] *Trans. Med.-Leg. Soc.*, vol. 19, 1925.
[4] *Lancet*, 1861, 2, p. 169.
[5] *Ibid.*, 1863, 1, p. 54.

was sensible, and while talking was suddenly seized with the usual symptoms. She had only three paroxysms. There is an instance reported in which a person is said to have recovered from a dose of seven grains of strychnine,[1] and one in which recovery took place under treatment after the taking of twenty grains of sulphate of strychnine.[2] In reference to this alleged recovery from large doses, it may be a question whether the strychnine was not mixed with some other substance, whereby its poisonous properties were weakened. Instances of recovery from doses above one or two grains must be regarded as exceptional.

With respect to *nux vomica*, three grains of the alcoholic extract have destroyed life. The smallest fatal dose of the powder, was in a case reported by Hoffmann, and quoted by Christison, also by Traill.[3] *Thirty grains* of the powder, given in two doses of fifteen grains each, proved fatal. The poison was given by mistake for bark to a patient labouring under quartan fever. This is about equivalent to the weight of one full-sized seed, and to only one-third of a grain of strychnine. The dose of nux vomica required to destroy life became of some importance in *R.* v. *Wren*.[4] The prisoner was convicted of an attempt to administer this poison in milk ; the quantity separated from the milk amounted to forty-seven grains. The intense bitterness which the nux vomica gave to the milk led to detection.

Duration. The time at which the symptoms commence appears from the recorded cases to be subject to great variation. In poisoning by nux vomica the symptoms generally appear more slowly than in poisoning by strychnine. Until they set in the patient is capable of walking, talking, and going through his or her usual occupations. In one case a man swallowed about 300 grains of nux vomica, and no symptoms appeared for two hours. He then died rapidly in a violent convulsive fit. On an average in poisoning by strychnine the symptoms appear with in from five to twenty minutes. In one case convulsions came on in five minutes.[5] In two cases at least, an hour has elapsed.[6]

The longest interval recorded was in the following case : A boy, *æt.* 12, swallowed a pill containing three grains of strychnine. No symptoms appeared for *three hours* ; they then set in in the usual way, and death took place in ten minutes. It was proved that the pill taken contained three grains of strychnine, with mucilage. The pills had been prepared eight months previously for the purpose of poisoning dogs ; hence they were hard, and would undergo only a slow solution in the stomach.[7]

The form in which the poison is administered or applied has a considerable influence on the time at whi h the symptoms commence. Thus when strychnine is given in pills especially if, as in the above case, they are hard, the symptoms are much longer in appearing than when the poison is taken in solution. When strychnine is given in solution the symptoms soon appear, and death takes place rapidly. This fact

[1] *Med. Gaz.*, vol. 41, p. 305.
[2] *B.M.J.*, 1892, 2, p. 179.
[3] " Outlines," p. 137.
[4] Winchester Spring Ass., 1851.
[5] *Ann. d'Hyg.*, 1861, 1, 133.
[6] *Lancet*, 1850, 2, p. 259.
[7] *Ibid.*, 1861, 2, p. 480.

connected with the absorption of this poison has been ignored. Palmer gave to the deceased, Cook, two pills supposed to contain strychnine. No symptoms were observed for an hour and a quarter. More than one expert deposed that this interval rendered it impossible that the symptoms could have been caused by strychnine. The above-mentioned cases will show that this opinion was in conflict with ascertained facts.[1]

If the poison is given hypodermically or applied to an ulcerated or diseased surface, or even to a healthy mucous surface, absorption may take place rapidly, and the interval for the production of symptoms is then proportionately short.

Period at which Death takes place. Death usually occurs after four or five convulsions and usually within about an hour after taking the drug, and it is exceptional to live for more than two hours after a fatal dose. Many cases have been reported, however, in which death occurred within less than an hour and also in which life has been prolonged for several hours. In the latter there has usually been delay in absorption of the drug. In the case of Warner the symptoms commenced within five minutes, and death took place within about eighteen minutes. On the other hand, in the case of Cook the symptoms did not commence until fifty-five minutes after the poison was taken, but the case terminated fatally within twenty minutes after their commencement.

Gray refers to a case which proved fatal within five minutes.[2] One of the longest cases of duration was that of an adult, who died within *six hours* from a dose of three grains of strychnine.[3] Some years ago Dr. F. J. Smith had an accidental case under his care (it was doubtless strychnine poisoning originally); the girl recovered from the definite symptoms of the strychnine, but died *two days* later, apparently from exhaustion.[4] In 1876, a case was tried[5] in which the prisoner was convicted of the murder by means of strychnine of a woman with whom he cohabited. The poison was administered in the form of vermin killer, given in decoction of sarsaparilla. The woman lived about five hours and three-quarters after taking the poison.[6] Clover survived about six hours. In 1893, at Warialda, in Australia, a man took on an empty stomach about seven or eight grains of strychnine; he lived for nine clear hours. Symptoms began within fifteen minutes. Under treatment by washing out the stomach and administering chloral and chloroform, convulsions subsided for over four hours, and then returned and proved fatal at the time stated. The case was recorded by Dr. J. T. Henry, medical officer of the Warialda Hospital, where the victim was treated, and he remarks that the usual assumption that, if a patient lives four hours after poisoning by strychnine he is practically safe, was not borne out in the case.

In poisoning by *nux vomica*, death usually occurs within two hours; but Christison mentions a case in which a man died within *fifteen minutes* after taking a dose.[7] This is probably the shortest period recorded.

Symptoms. At a variable interval after taking either nux vomica or strychnine in a poisonous dose, the patient experiences a sense of

[1] *R.* v. *Palmer*, C. C. C., 1856.
[2] "Strychnine," 1872, p. 55.
[3] Guy's Hosp. Rep., 1857, p. 483.
[4] Lond. Hosp. Case Records, 1894.
[5] *R.* v. *Silas* Barlow, alias *Silas Smith*, C. C. C., November, 1876.
[6] *Pharm. Jour.*, December 2nd, 1876, p. 467.
[7] *Op. cit.*, p. 898.

uneasiness and restlessness, accompanied by a feeling of suffocation, and not infrequently by a sense of impending calamity or death. There is a shuddering or a trembling of the whole frame, with twitchings and jerkings of the head and limbs. Tetanic convulsions then commence suddenly with great violence, and nearly all the muscles of the body are simultaneously affected. The limbs are stretched out involuntarily, the hands are clenched, the head after some convulsive jerkings is bent backwards, and the whole of the body becomes as stiff as a board. As the convulsions increase in frequency and severity the body assumes a bow-like form (opisthotonos), being arched in the back and resting on the head and heels. The head is firmly bent backwards, and the soles of the feet are incurved, or arched and everted, the legs sometimes separated. The abdomen is hard and tense, and the chest spasmodically fixed, so that respiration is arrested. The face assumes a dusky, livid, or congested appearance, with a drawn, wild, or anxious aspect; the eyeballs are prominent and staring, and the lips are livid. The intellect is clear, and the sufferings during this violent spasm of the voluntary muscles are severe. The patient in vain seeks for relief in gasping for air, and in requiring to be turned over, moved, or held. The muscles of the lower jaw are generally the last to be affected by this poison. The jaw is not always fixed during a paroxysm. The patient can frequently speak and swallow, and great thirst has been observed among the symptoms. In some cases of poisoning by nux vomica, the jaw has been fixed by muscular spasm, which has come on suddenly in full intensity with tetanic spasms in other muscles. The sudden and universal convulsion affecting the voluntary muscles has sometimes been so violent that the patient has been jerked off the bed. After an interval of half a minute to one or two minutes the convulsions subside; there is an intermission. The patient feels exhausted, and is sometimes bathed in perspiration. It has been noticed in some of these cases that the pupils during the paroxysms are dilated, while in the intermission they are contracted. The pulse during the spasms is so quick that it can scarcely be counted. Slight causes, such as an attempt to move, a sudden noise, a draught of air, or gently touching the patient, will frequently bring on a recurrence of the convulsions. In cases likely to prove fatal they rapidly succeed each other, and increase in severity and duration, until at length the patient dies utterly exhausted. The tetanic symptoms produced by strychnine, when once clearly established, progress rapidly either to death or recovery. The patient is conscious, and his mind is commonly clear to the last. He has a strong apprehension of death. Generally speaking, within an hour or two from the commencement of the symptoms the person either dies or recovers, according to the severity of the paroxysms and the strength of his constitution. The temperature of the body often rises above the normal after death.

Strychnine poisoning may in some respects resemble tetanus. The following table shows the chief differences :—

Strychnine.	*Tetanus.*
1. Sudden onset in previous good health.	1. Gradual onset with some premonitory symptoms of illness.
2. Does not commence in, nor especially affect, the jaw.	2. Usually commences in, and especially affects, the lower jaw.

Strychnine.	*Tetanus.*
3. Relaxation between the fits is quite complete.	3. Relaxation between the spasms never quite complete. Some residual stiffness is invariable.
4. Steadily worse or steadily better.	4. Progress rarely steady in either direction unless case is very severe or very mild.

The spasms due to spinal meningitis may be diagnosed by the history, the high temperature and from spinal punctures. The spasms of pilepsy are distinguished from those of strychnine by the loss of consciousness in the former. Hysterical states are distinguished by the difference in onset usually but occasionally may be indistinguishable from strychnine poisoning, but if there is any difficulty then treat as for strychnine poisoning.

Treatment. It is most important to control the tetanic spasms before attempting any other treatment otherwise the patient will die. The patient must be kept absolutely quiet in a darkened room, free from draughts or sudden noises and movements. It may be necessary to put the patient lightly under chloroform but as soon as possible a barbiturate, such as sodium pentobarbital (3–5 grains) or sodium amytal (5–10 grains), should be administered slowly by ntravenous injection watching the effect. If the convulsions cease, further doses may be given orally.[1] Having controlled the spasms, wash out the stomach with warm water in which animal charcoal is suspended, or with 0·1 per cent. potassium permanganate solution. After washing out the contents a suspension of charcoal should be left in the stomach. If the stomach cannot be washed out an injection of apomorphine $\frac{1}{10}$ grain should be given to procure emesis or one of the ordinary emetics given by the mouth. Spontaneous vomiting does not usually occur. After emptying the stomach the patient may be kept under the influence of a barbiturate. If there is any respiratory difficulty artificial respirations may have to be adopted. The patient must be kept under observation for several hours. Chloral hydrate has been used as an antagonist to strychnine. A man swallowed two three-penny packets of Battle's "Vermin Killer," which produced typical symptoms of strychnine poisoning; the patient did not vomit, nor was the stomach emptied. Twenty grains of chloral hydrate dissolved in water were injected subcutaneously, followed by a second dose of twenty grains, and subsequently by ten grains more; twenty grains were also given by the mouth as soon as the patient could swallow; recovery took place.[2]

Post-mortem Appearances. There are no specific appearances found in strychnine poisoning. The ordinary signs of asphyxia are observed and there may be peticheal hæmorrhages in some of the viscera. The brain and spinal cord must be examined carefully to exclude any conditions which might cause convulsions.

Rigidity of the muscles occurs more rapidly than in normal death but is not prolonged unduly. In rabbits poisoned by strychnine, the author observed the body of one to remain perfectly rigid for a week.

[1] Travell and Gold, *Jour. Pharm. and Exp. Therap.*, 51, 129 (1934).
[2] *Lancet*, 1889.

while another had lost all rigidity and had begun to putrefy after thirty-six hours. It is not any special influence of the poison on the muscles, but the mode in which it operates on the system, that determines the commencement and duration of rigidity in the dead body.

The rise of temperature after death in strychnine poisoning may cause the body to be warm for an unusual length of time.

Analysis. Strychnine is rapidly absorbed mainly from the small intestine but also from the stomach. It appears to be in great part detoxicated by the liver and also by the muscular tissues. A prop rtion is excreted in the urine. It is as a rule readily discovered by analysis even after long periods. Persons have died from strychnine, and no trace of the poison has been found in the body. In a case of poisoning by this alkaloid, which was the subject of a trial for murder in 1861, Reese made separate analyses of the contents of the stomach and the contents of the intestines, as well as of the tissues, and each one of these was repeated to avoid error. Yet there was no evidence of the presence of strychnine by the bitter taste of the final extract, nor by the colour tests. In Reese's case the quantity taken was unknown, the woman lived five or six hours, and the body was not examined until six weeks after death. A small but fatal dose, and the duration of the case, will sufficiently account for the negative results without resorting to any other hypothesis.

The following case shows the rapidity with which the poison may be diffused and deposited in the tissues when a large dose has been taken :—

A strong, healthy man, *æt.* 43, placed upon his dry tongue, at 10 p.m., a powder which contained six grains of Dover's powder and five grains of strychnine (dispensed by mistake for five grains of James's powder). He complained of a bitter taste, asked for an orange, and found, on sucking this, that it increased the bitterness ; the acid juices of the orange dissolved the strychnine, and thus favoured its early absorption. In fifteen minutes he went to bed. Twitchings of the muscles then came on, and the patient, from previous experience in taking strychnine as a medicine, was fully aware of the cause of the symptoms. He complained of his bowels being drawn up ; he drew his knees up as if to his mouth, gave a yell, seized a friend who was standing by, and became apparently unconscious (exhausted) for about five minutes. He then revived, but in a few minutes was again seized with a violent convulsion of a tetanic character, and he died in two or three minutes afterwards, a little over half an hour after taking the powder. Owing to a spasmodic closure of the jaws, he was able to speak only for a few minutes at a time ; he was rational, but seemed to be in great terror.

Edwards found strychnine in the stomach, the quantity being estimated at about one grain. He also found the poison in the tongue and the liver. He sent to the author a portion of the liver, one kidney, and six ounces of blood. They were in a putrescent state, and when examined about three months after death, eight ounces of the liver yielded prismatic crystals of strychnine, producing the usual colour reactions. The quantity of strychnine thus obtained was small. The kidney and the blood did not give results on which any reliance could be placed.

In Palmer's or rather Cook's case there was such gross and culpable mismanagement of the whole proceedings that it affords no corroboration or disproof of the non-detection of strychnine.

For a more complete history of the *medical* facts in the memorable case of Cook, *vide* "Poisoning by Strychnia," Guy's Hosp. Rep., 1856 ; *Pharm. Jour.*, July 1856, p. 6 (from the pen of the late Jacob Bell).

A most able legal analysis of it was published by Stephen in his "General View of the Criminal Law of England" (1863, p. 357). Of the foreign reports one by Tardieu, in the *Ann. d'Hyg.*, for 1856, 2, 371, and 1857, 1, 132, and the other by Casper, in Horn's *Vierteljahrsschr. für Ger. Med.*, etc., 1864, 2, p. 1, are the most correct in their medical and medico-legal details.

Sir Thomas Stevenson believed that much smaller quantities of strychnine might be detected when mixed with organic matters than was considered possible by Taylor; and that with improved methods of analysis strychnine could not fail to be detected in the body in any case of poisoning by this alkaloid proving fatal within a couple of hours. As a rule it may be readily detected in the urine during the course of a case of strychnine poisoning, and also when the alkaloid is being given in ordinary medicinal doses. Strychnine resists decomposition to a greater extent than the other common alkaloids, and hence, when given in large doses, is often detected in the exhumed bodies of animals. Sir Thomas Stevenson extracted one-sixteenth grain from two pounds of the exhumed viscera of a woman (Matilda Clover) more than six months after burial, although the deceased had survived the administration of the poison for six hours. There was, so far as he was aware, no other recorded case of the detection of strychnine in this country after exhumation, though two cases are recorded on the Continent of its detection after exhumation of human remains.[1]

Powdered nux vomica yields to water and alcohol, strychnine, brucine, igasuric acid, and some common vegetable principles, the strychnine varying from 0·28 to 1·8 per cent. Heated on platinum foil, it burns with a yellow smoky flame. Nitric acid turns it a dark orange-red colour, which is destroyed by stannous chloride. These properties are sufficient to distinguish it from various medicinal powders which it resembles. In one case of poisoning by this substance[2] a quantity of guaiacum powder was mixed with the nux vomica. This so completely changed the action of nitric acid as in the first instance to create some difficulty in identifying the substance. The analyst must be prepared for these admixtures or adulterations.

The aqueous *infusion* or *decoction* is reddened by nitric acid, and is freely precipitated by tincture of galls. Ferric sulphate gives with it an olive-green tint. The fine silky fibres or hairs which cover the surface of the seed may be obtained by washing the residue of the powder in the stomach, where it remains unaltered, or the sediment of any liquid with which the nux vomica may have been mixed. They present a characteristic appearance under the microscope. Strychnine may be extracted from nux vomica by the process described on p. 763.

There is one caution to be given in reference to the examination of the stomach. As death is commonly rapid, and there is no vomiting, the colouring matter, either soot or Prussian blue, should always be sought for in the stomach. Strychnine may or may not be found according to the amount swallowed and the degree to which absorption has gone on during life.

Strychnine. This alkaloid may be readily obtained in crystalline form from an alkaline solution. The crystals are very small, and their

[1] *R.* v. *Neill*, C. C. C., October 1892.
[2] *R.* v. *Wren*.

form is subject to great variation, according to the strength of the solution, the rapidity or slowness of evaporation, the presence of foreign matters, etc. They are commonly seen in octahedra, sometimes lengthened into prisms, bevelled at the ends, and crossing each other at angles of 60°. There are as many as six or eight varieties of crystals, so that too much importance must not be attached to their form. When strychnine is procured from the solutions of its salts by the addition of ammonia, it is usually deposited in long slender prisms.

Tests. (1) Strychnine ($C_{21}H_{22}N_2O_2$) is a white crystalline powder with an intensely bitter taste even in solution at a dilution of 1 in 30,000. (2) When strongly heated on platinum, it melts, and burns like a resin with a black smoky flame ; in a narrow tube it yields ammonia. (3) Sulphuric acid produces no apparent change in it ; but when to the mixture a small crystal of either bichromate of potassium, ferricyanide of potassium, or a small quantity of black oxide of manganese (manganese dioxide), cerium peroxide or peroxide of lead is added, a series of beautiful blue, violet, and purple colours appear, which pass rapidly to a light red tint, fading to yellow. Among these substances, black oxide of manganese will be found preferable. By reason of its insolubility, it imparts no colour to the liquid if strychnine is absent ; and if the alkaloid is present it slowly brings out the colours, so that there is time to make full observation. The hydrated or precipitated oxide may be used in place of the anhydrous compound. Cerium peroxide gives slow reactions in the fading purple test and is therefore more suitable for the detection of small quantities. The same may be said of Mandelin's reagent, which gives a similar reaction. Letheby suggested the use of a small galvanic battery for the production of the coloured reaction. In this case sulphuric acid only is required. It presents no practical advantage over the use of oxide of manganese. Gelsemine is the only alkaloid which gives a similar reaction, but the colour in this case is first red, then violet, and finally green. A large excess of bru ine or morphine interferes with the reaction. The effect of brucine, however, can be avoided by dissolving the alkaloids in sulphuric acid, warming with a trace of nitric acid, and completing the test only when the red colour produced by the brucine has faded to yellow. In this way the brucine is destroyed. (4) It is not perceptibly dissolved by cold water, requiring 7,000 parts for its solution. (5) It is easily dissolved by acids, and is precipitated from its concentrated solutions by potash or ammonia, in both of which it is insoluble. (6) Strong nitric acid imparts to commercial strychnine a pale reddish colour, owing to the presence of brucine. (7) Sulphomolybdic and iodic acids produce no change of colour in strychnine, and thus distinguish it from morphine. (8) Potassium ferrocyanide, added to a solution of a strychnine salt, gives a yellowish-white crystalline precipitate, sparingly soluble in cold acidified water. Brucine gives no precipitate except from very concentrated solutions. (9) 1 c.c. of a dilute solution of a strychnine salt (less than 0·1 per cent.) is mixed with 2 c.c. of hydrochloric acid and 1 gram of granulated zinc. After two or three minutes the mixture is boiled, cooled, and the liquid carefully poured, to form a separate layer, on 2 c.c. of concentrated sulphuric acid in a test-tube. In presence of strychnine a rose-red zone appears at the junction of the layers and gradually spreads through the liquid. The colour is not destroyed by boiling but is destroyed by potassium

thiocyanate, or ammonia, or sodium hydrogen sulphate in excess. Veratrine gives a similar colour, turned to a dirty yellow by boiling. (10) If a solution of strychnine is administered hypodermically to a frog, a period of uneasiness and respiratory difficulty is succeeded by tetanic tremors and then by tetanic convulsions. These occur intermittently, with periods of rest, and end in death if the dose is large enough. As little as 0·06 mgrm. can be detected in this way. The solution for injection should be as nearly neutral as possible.

The *salts* of strychnine are readily soluble in water, and these solutions are precipitated by the usual group reagents for alkaloids. The alkalies and alkaline carbonates, if diluted, precipitate the alkaloid in slender prisms. Sulphocyanide of potassium and chromate of potassium give well-defined prismatic crystals. In the last case the crystals are stellated and of a yellow colour. Chromate of strychnine may also be produced by adding bichromate of potassium to a dialysed liquid containing strychnine. On draining the crystals, drying them, and adding sulphuric acid, the colour reactions are at once brought out. Picric acid is an even more delicate precipitant of a solution of strychnine. It gives small tufts or groups of stellated crystals. Sulphocyanide of strychnine in crystals may be produced in solutions containing not less than one seven-thousandth part of strychnine. Filhol recommends chlorine and auric chloride as delicate precipitants of solutions of strychnine, taking care that there is no alcohol in the liquid to be tested. Auric chloride slowly precipitates, in a crystalline form, even the six hundred and fiftieth part of a grain of strychnine. When these precipitates, drained of water, are treated with concentrated sulphuric acid, they are dissolved, and to this mixture a crystal of bichromate of potassium may be added to bring out the blue coloration peculiar to strychnine.

Strychnine has been fatally mistaken for santonin,[1] salicin, and jalapin, and has thus caused death on several occasions. Jalapin does not crystallise, and the crystalline forms of santonin and salicin are very different from those of strychnine.

These two vegetable principles differ from strychnine in their properties. When heated in closed tubes, they give off *acid* vapours. Salicin is soluble in water. Santonin is not soluble in water, but is dissolved by alcohol. Tannic acid and potassio-mercuric iodide do not precipitate the solutions, while they readily precipitate those of strychnine. Nitric acid has no effect upon either, while sulphuric acid, which does not change santonin, gives a pink colour to salicin. The crystals of santonin closely resemble, in microscopical appearance, those of salicin, but they are distinguished from salicin and from alkaloids by acquiring a brilliant yellow colour on exposure to sunlight without undergoing any change of form.

From *organic mixtures* the alkaloid is separated by the process on p. 265.

By this method strychnine may be detected in the organs of a person who has died from this poison, although the organs are in a highly putrescent state. The process of dialysis will allow of the separation of strychnine, when combined with acids and in a state of solution, from blood, mucus, and other viscid organic matters found in the stomach

[1] *Lancet*, 1870, 1, p. 598.

On three or four occasions it has been detected in exhumed bodies, in the case of Clover six months after burial. In all cases the physiological test should be used to supplement the chemical tests for strychnine. A frog may be employed for this purpose.

Strychnine in Organic Solids. From vermin killers the strychnine may generally be readily separated by means of alcohol, and obtained in a crystalline form for the application of tests. If the vermin killer is coloured with Prussian blue, one or two drops of sulphuric acid will remove the colour from the separated alkaloids, and black oxide of manganese may be added. The colour reactions are then as well marked as with pure strychnine.

The alkaloids strychnine and brucine may be detected in the **powder of nux vomica** by the following process:—Digest the powder in a small quantity of dilute sulphuric acid on a boiling water-bath. The substance should be well stirred with the dilute acid, which, after a short time, completely carbonises it. The mass is heated to dryness, then treated with a small quantity of distilled water and filtered, by which an acid liquid of a pale sherry colour is obtained. On neutralising this liquid with potash or ammonia, and agitating it with twice its volume of ether, the strychnine is separated, and may be obtained crystallised by the evaporation of the ethereal solution. The strychnine may also be obtained by dialysis. Ten grains of nux vomica, equal to three-tenths of a grain of strychnine, give satisfactory results. Prismatic crystals are pro u ed which give the appropriate reactions with the colour tests. Brucine may also be detected by the action of nitric acid on the crystals. Brucine is much more soluble in alcohol than strychnine. If, therefore, the mixed alkaloids are dissolved in hot alcohol, they may conveniently be separated by fractional crystallisation.

In cases in which the poison is contained in pills or powders having much organic matter soluble in alcohol, it will be advisable to employ either dilute or concentrated sulphuric acid. It is a remarkable fact that strychnine itself is not acted on in the same degree by sulphuric acid as ordinary organic substances, or even other poisonous alkaloids.

Analysis of Brucine ($C_{23}H_{26}N_2O_4$). An alkaloid generally associated with strychnine. It is contained in the seeds of nux vomica, and more abundantly in the bark of the tree. It is not so powerful a poison as strychnine, but the symptoms which it produces are similar. It is considered to have from one-twelfth to one-fortieth the strength of strychnine. It is not affected by the colour tests employed for the detection of strychnine, but it acquires an intense red colour on the addition of nitric acid, and this is changed to a violet on the addition of stannous chloride (the colour is destroyed by excess of nitric acid or of stannous chloride). It is more soluble in water than strychnine, and has a similar bitter taste. Hydrochloric and iodic acids produce in it no change, either in the cold or when heated. Sulphuric acid usually gives to it a pink-red colour without carbonising it, owing to the presence of a trace of nitric acid in the acid; sulphomolybdic acid does the same, but the red colour changes to greenish brown, and ultimately a blue black. The sulphate of brucine crystallises in well-defined prisms truncated at the ends. They are larger and longer than the prisms of strychnine.

Cases. The following personal narrative of an accidental case of strychnine poisoning is so interesting as to deserve nearly verbatim report. It is taken from the *South African Med. Jour.*, April 1895, pp. 341 *et seq.*, and is from the pen of Dr. W. T. Harris, himself the victim :—

"In January 1893, it happened that I had for a few weeks been in the habit of taking an occasional dose of one of our stock dispensary mixtures—a tonic containing, amongst other things, a fair dose of strychnine. The weather was very sultry, the work very onerous, as it always is in the first few weeks of the year, and I was hourly expecting a cablegram from home to announce a bereavement which can only occur once in a lifetime, and which in fact did come four days later. It was therefore not because of any real illness, but from being anxious and below par, that on the morning of Tuesday, January 10th, I went into the dispensary before the dispenser had arrived, to take a dose of the tonic I have alluded to. It is kept in a concentrated form, the whole bottle containing five drachms of the liq. strychninæ B.P., and each ounce of the diluted mixture five minims. Somewhat carelessly I poured out sufficient to make an ounce and a half, and filling up the measure-glass with water, drank if off.

"I at once noticed a much more intensely bitter taste than was usual; for although this characteristic of the drug may be detected in very dilute solutions, it seemed increased tenfold, as indeed it was almost, as I shall presently show. I immediately asked the porter if he knew when the mixture had been made up, and he replied that it had been done on the previous day, but as yet none had been dispensed from the bottle.

"I did not quite know what to do, and my first impulse was to take an emetic; but, as the swallowing of saliva lessened the bitter taste every minute that I hesitated, I persuaded myself that the difference might be only fancy.

"I had made a good breakfast, and was loath to sacrifice my mutton-chop and upset my stomach, only to be laughed at; for how could a large stock-bottle be made up so improperly that an ordinary dose would do me harm? And was it not ready to be dispensed for a number of other people? I shook off my fancies therefore, and going into the consulting-room, rang the bell for out-patients and went on with the morning's work.

"Fifteen minutes elapsed, and I began to feel very restless. An indescribable nervous sensation came over me, as if there were rope pulleys running down to my extremities, which were gradually being drawn tight. I had to make an effort to prevent my mouth closing too soon as I spoke, and to dig my pen into the paper and write thick, as if to form a fulcrum over which to lever my hand along the pages, while a contra-force in my arm strove to dash the pen to the floor.

"Fortunately there were but few patients to see that morning, and I had just finished them, when, at a little before eleven o'clock, Dr. Considine, who was visiting surgeon for the day, came in. I at once told him that I felt very strange, and feared I had taken an overdose of this strychnine mixture. He laughed and said I was nervous, knowing that we both had taken the same medicine often with impunity. He then commenced talking on some topic in which we were usually interested, when I broke in abruptly, saying, 'I feel I cannot sit still and talk; let us go round the wards.'

"We started through the principal male ward, which is a daily routine, and one always of interest, and generally of pleasure. But the simple round on that particular day seemed then in fact, what it still appears to memory, a dreadful nightmare. My limbs were throwing off the control of will, and moved erratically; when I wished to go on, my legs stopped, and when by a violent effort I forced them to proceed, I could not pull up to a standstill without walking against a bed to steady myself. What I said or did I cannot remember, but I managed to get along somehow, though feeling as if head, hands, and legs belonged not to me, but to three separate individuals, like a mechanical doll that has had all its limbs jerked with each pull of the string. At length we returned to the top of the ward, when, feeling a paroxysm down my back, I said to Dr. Considine, 'I am really very ill. I feel sure I am suffering from strychnine poisoning.'

* * * *

"I had taken six-tenths of a grain."

[Then follows a most graphic description of his thoughts and feelings as a condemned man. He said,] "Shall I have an emetic?" and Dr. Considine said, "No, it is too late; take sixty grains of chloral." "Now go," said the doctor, "to the ophthalmic room, and smoke hard, if you can manage to."

"I turned to go. 'You'll come soon; do not leave me for long,' I said. 'I'll come immediately, and not leave you, however long it is, till you are better.' The words imbued me with new courage, though, as he told me afterwards, he feared the worst, and only stayed to get chloroform, morphia, and a hypodermic syringe, should they be wanted.

"I got down the passage, lay on the couch, and tried to smoke, but there was no rest possible: it was like lying on the felt floor of a Turkish bath. As one flinches there from the heat of contact with surrounding objects, so here every touch sent a tetanic convulsion through me. I could not rest; should I get worse and have opisthotonos? Would the chloral stop, or only stay, the action of the poison? Was it to be a reprieve, or only a respite? I started to my feet and got over to the book-case, to see what Taylor's 'Jurisprudence' said as to the oncoming of symptoms and the period of danger.

"The book was not there, and I remembered that I had taken it over to my residence. How was I to get across the garden to my study, sixty or seventy yards away? What should I do if I met any one, how afford an explanation with every muscle on the rack, and feeling unable to articulate? I could only hope to get in and out unobserved, for I felt that not only would any attempt to explain bring on a paroxysm, but that I should cause the greatest alarm by my appearance. I started, and how I steered myself across is a problem still. I ran in jerks and jumps, just as a drunken man makes a dash from one lamp-post to another.

"I regained the room in the hospital, and, steadying myself between couch and table, turned to the accounts of strychnine poisoning, feeling a 'trembling of the whole frame' and 'impending suffocation' as I hurriedly glanced at those very words, so well describing my own symptoms. The accounts were conflicting:—'A man swallowed three hundred grains of nux vomica, and no symptoms appeared for two hours; he died speedily in a convulsive fit'; again, 'In the case of Dr. Warner, half a grain of strychnine destroyed life; the symptoms commenced in five minutes, and he died in twenty minutes'; further on: 'The longest period at which death has occurred was six hours after the administration of the poison,' and I thought that, like me, he might have taken it on a full meal. There was no comfort so far, but at last my eye fixed on this: 'In fatal cases death generally takes place within two hours.' To that I pinned my faith, for it was nearly twelve o'clock, and every moment was a step towards safety.

"I was now able to lie down, for the chloral was certainly taking effect, not as a hypnotic, for I was never more wide awake, but I could feel it 'coursing through the narrow straits and alleys of the body,' with a gentle glow, and the spasms were abating.

"Dr. Considine, who had been coming in and out, now settled down beside me, his cheery words being an important factor in tiding me over the next hour. He did everything to divert attention from myself, relating how he had once taken an overdose of strychnine, and had used tobacco as the only antidote available. The question of more chloral was considered, but no more was given, as I had had a large dose, which he rightly thought would prove sufficient; and more was at hand at any moment had acute symptoms returned.

"As is usual in these cases when once the poison is eliminated, I felt but little subsequent effect beyond some weariness after the shock.

"I have now come to the end of my narrative, in which I have endeavoured to portray faithfully the sensations, mental and bodily, that I went through. That I did not get worse, and actually recovered after only the one large dose of chloral, I attribute to my generally good constitution, to the fact that I had habituated my system somewhat to the action of strychnine by having taken for a week previously medicinal doses of it two or three times a day, and to the prompt administration of the antidote at the critical moment, when the symptoms were coming to a climax."

The following case, reported in the *B.M.J.*, 1894, 1, p. 300, is of interest as showing the stability of strychnine in solution. It is reported by Dr. Percy T. Adams :—

" Recently I attended a strong, healthy man of twenty-one years of age who died of strychnine poisoning. The points of interest in the case were : (1) Quantity of drug taken, thirteen and a half to eighteen grains, in the readily assimilable form of liq. strychninæ, B.P. ; (2) relatively slow speed (namely, fifteen to twenty minutes) at which so large a dose killed when taken upon an empty stomach, for the man drank it directly out of the bottle before breakfast. This solution had been kept often exposed to light for probably six years, but was apparently unaltered as regards its potency."

The following is reported by Dr. S. H. D. Hale, of Southsea, in the *B.M.J.*, 1899, 2, p. 10 :—

" I was called in July, 1898, at 2 p.m., to see a woman who was reported to have fallen and injured her head. She was lying semi-conscious on the floor, with a large but not dangerous scalp wound.

" On examination it was at once seen that the injury to her head was not the cause of her symptoms. It transpired that she had swallowed six drachms of the tincture of nux vomica in mistake for another drug. She quickly developed symptoms of strychnine poisoning. Exactly two hours after swallowing the fatal dose she died, during the third attack of general convulsions, and was not resuscitated after half an hour's artificial respiration.

" It was found quite impossible to pass the tube of the stomach-pump, as any attempt to do so at once brought on tetanic spasms ; and on administering chloroform she developed such dangerous symptoms of syncope that this had to be abandoned. Accordingly one-eighth of a grain of apomorphine was injected subcutaneously, but this had no effect whatever in producing vomiting, the dose being repeated in half an hour with a similar negative result. Two doses of chloral hydrate one drachm each were given by the mouth at intervals of half an hour, and during the three attacks of general convulsions a drachm of ether was injected subcutaneously, and hot flannels were applied to the precordial region.

" The chief points of interest about this case seem to be—(1) that the fatal dose was three-quarters of a grain of strychnine ; (2) the comparatively rapid action of the dose ; (3) that the pupils were widely dilated, and there was loss of consciousness on three occasions ; (4) the failure of apomorphine to produce vomiting ; and (5) the dangerous symptoms induced by the exhibition of chloroform."

For a case of severe vesical spasm following four minim doses of the B.P. liq. strych., *vide B.M.J.*, 1895, 1, p. 135.

In 1898, a trial for strychnine poisoning took place which is thus recorded and commented on by the *Lancet*, 1898, 1, p. 1628.[1]

" On June 6th, the trial of Walter Horsford for the murder of his cousin, Annie Holmes, a widow, was concluded. After five days' investigation at the Huntingdon Assizes, before Mr. Justice Hawkins, the prisoner was convicted and sentenced to death. The facts of the case were briefly as follows : Horsford, who had recently married, had received an intimation from Mrs. Holmes that she believed herself *enceinte*, with the implication that he was the author of her trouble. In his reply he repudiated liability on the ground that she had received monetary consideration, and expressed a wish that no more letters should be sent to him, as he did not wish his wife to know of the relations which had existed between them. In December last Horsford purchased at a chemist's a large quantity of arsenic, and ninety grains of strychnine, alleging that he wanted them to exterminate vermin. One day, early in January, Mrs. Holmes, who had been in her usual health, retired to bed, taking with her a glass of water, an unusual circumstance. Shortly afterwards her daughter, aged about thirteen years, found her mother in great agony. The neighbours were called in and Dr. Joseph Herbert Anderson, acting as assistant to a local practitioner, was fetched. He found deceased suffering from symptoms of strychnine poisoning and administered antidotal sedatives. Treatment proved of no avail, and in a short time the unfortunate woman expired.

[1] *R.* v. *Horsford.*

" At the inquest, Horsford, in his evidence, denied that he had had immoral relations with his cousin, or that he had visited her, or that he had sent anything to her. These statements were demonstrably contrary to fact, for he had been twice to see her, and there was the letter above referred to. He was arrested on a charge of perjury and was subsequently indicted for wilful murder. After Mrs. Holmes's death some letters were found beneath the mattress of the bed, together with two papers, one of which, though nearly empty, contained a little strychnine, whilst the other held thirty-five grains of the alkaloid. On one of the packets was written ' One dose take as told,' and also the significant expression ' It is quite harmless.' The analysis of the viscera and their contents revealed the presence of a large quantity of strychnine. From the characteristic symptoms and the chemical analysis, there could be no shadow of doubt that the deceased had died from strychnine poisoning. The questions to be determined were : Was the alkaloid taken by accident, or was it administered with a felonious intent ; and if the latter, who was the guilty person ? Although strict inquiries were made, none other than Horsford likely to be known to Mrs. Holmes, and to be interested in her affairs, was found to have purchased strychnine from the chemists for some distance around. Fortunately there was no difficulty in comparing Horsford's known handwriting with the direction on the packet containing the powder. The likeness between the two was so striking that it scarcely needed an expert to appreciate it. Now, whether Horsford caused the poison to be administered with the intent to procure abortion, a proposition purely visionary, or to compass death mattered not. The *post-mortem* examination showed that Mrs. Holmes had not been pregnant within a short period of her death. But even this mistake as to her condition would not have diminished the criminal responsibility, since the Act in considering the penalties for the procuring of abortion distinctly says ' whether the woman be pregnant or not.' The chief contentions for the defence were (1) that some other person than Horsford might have been the guilty party ; (2) that the strychnine found in the papers might have been put under the bed subsequently to the death of Mrs. Holmes ; and (3) that it was not likely that Horsford, ' who only valued Mrs. Holmes's body at half a crown,' a sum he acknowledged to have given her, would sacrifice her body and soul at the risk of losing his own life. As regards the first, it may be remarked that there was not the slightest testimony that any other person than Horsford had any reason for taking her life. The other points are too puerile to merit discussion."

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Poisoning by Taxus baccata, or Yew (Coniferæ)

Source and Method of Occurrence. The yew tree (*Taxus baccata*) is indigenous in England, and in the days of bows and arrows was largely planted, so that it is now a very common tree. Poisoning by it is almost entirely a matter of accident, and is much commoner in cattle than in human beings. The red pulp of the fruit has a sweetish pleasant mucilaginous taste, and is innocuous, but the green seed inside the pulp is distinctly poisonous. It is now well ascertained that yew leaves and the hard central portion of the berries exert a specific poisonous action both on men and cattle. If animals recover from the primary effects, they are liable to die after several days from inflammation of the bowels. On one occasion Taylor examined the viscera of an ox which had died from the poisonous effects of yew leaves. There was much inflammation, and some parts of the intestines were gangrenous. Infusion of yew leaves, which is popularly called yew-tree tea, is sometimes used for the purpose of procuring abortion by ignorant midwives.

Toxicity and Fatal Dose. The toxicity of the plant is due to taxine, an alkaloid obtained from the berries and leaves. It is not official, but its dose[1] is given as one-hundredth to one-sixtieth of a grain. It is a dangerous narcotic poison.

There is no record of the exact minimal lethal dose, but a teaspoonful of the leaves has proved fatal.

A lunatic woman had been employed in preparing evergreen decorations. Nothing unusual was observed by the nurses in attendance until about 10.30 p.m. She had had some bread-and-cheese with the other patients, when in about five minutes she slipped off her chair almost helpless. Her countenance turned a dusky pallid hue, but there were no cerebral symptoms. She vomited a quantity of food mixed with a few bits of yew leaves. She soon passed into a state of collapse, and died in less than three hours from her first seizure. She retained her consciousness until a few minutes before she died, and admitted that she had eaten some little bits of yew, but she did not think anything of it. The broken leaflets in the vomited matters and the portions found in the stomach and bowels after death did not amount to a teaspoonful. Yew leaves may thus prove in small quantity a rapidly fatal poison.

For further points *vide B.M.J.*, 1899, 2, p. 1377.

Duration. Symptoms appear within an hour or less; and death or recovery ensues within eight or ten hours, but may be delayed for longer periods. In the non-fatal case described below the symptoms did not come on till the fourth day after taking a decoction of the plant.

Symptoms. The symptoms produced by the *leaves* and *berries* are fairly uniform in character; convulsions, insensibility, coma, dilated pupils, pallor, small pulse, and cold extremities, are the most prominent. Vomiting and purging are also observed among the symptoms. The subject of one case, a girl about five years of age, died in a comatose state in four hours after she had eaten the *berries*, and in another case

[1] Martindale, "Extra Pharm."

a boy, *æt.* 4 years, died nineteen days after taking the berries, obviously from severe inflammation of the bowels. The immediate symptoms in the boy were vomiting, purging, coma, convulsions, dilated pupils, hurried respiration, a small pulse, and a cold skin.[1] A tablespoonful of the fresh *leaves* was administered to three children of five, four, and three years of age as a vermifuge. Yawning and listlessness soon succeeded; the eldest vomited a little, and complained of pain in the abdomen, but the two younger children suffered no pain. They all died within a few hours of each other.

An insane person died from the poisonous effects of *yew leaves*. The patient was observed chewing the plant, and before the attendants had taken it from him he had succeeded in swallowing a portion of the masticated juice. He was soon afterwards suddenly seized with giddiness, prostration of strength, vomiting, coldness of the skin, spasms and irregular action of the heart. He died within fourteen hours. On inspection the stomach was found much distended; and it contained some yew leaves.[2]

Treatment. Empty the stomach, and counteract collapse by all general measures. There is no specific antidote known.

Post-mortem Appearances. There may be gastro-intestinal irritation but nothing characteristic.

Analysis. Fragments of the leaves or the berries may be found in the stomach. The yew and the savin are the only coniferous poisonous trees which are likely to require separation. The apex of the leaf of the yew is not so pointed as that of the savin, and the yew leaf does not possess the peculiar odour of savin when rubbed. Yew berries are seen in autumn; they are about the size of a pea, of a light red colour, dull on the surface, and translucent. They are open at the top, allowing a hard brown kernel to be seen. This is of an ovoid shape, and it forms the greater part of the fruit. The fine red skin contains a colourless and remarkable viscid or adhesive juice, which reddens litmus paper, and has a sweetish taste.

Taxine may be sought for by the ordinary process for obtaining alkaloids. It gives a rose-pink colour with concentrated sulphuric acid, which fades on dilution. With sulphuric acid and potassium bichromate it gives a bluish-purple colour; with Fröhde's reagent a deep violet colour; with nitric acid a red-violet solution turned brown by warming; and with hydrochloric acid a violet or violet-grey colour.

Cases. In 1902 a trial took place[3] before Mr. Justice Bucknill, in which the accused was charged with administering yew to a woman with intent to procure abortion. The accused was acquitted on the ground that she did not know that the girl was pregnant. The case is thus recorded by Dr. Barling:—

"A pan was filled with leaves and twigs of yew, filled with water, and boiled for several hours. A teacupful was taken three times a day. For three days nothing was noticed. On the fourth day the girl complained of 'pins and needles' in the epigastrium, followed by nausea and constant pain. On the seventh day she vomited. The pain got worse, and she had diarrhœa. At the end of a fortnight she could hardly stand, and one night became unconscious. The stuff was discontinued, and she rapidly recovered."

[1] See *Prov. Jour.*, November 29th, 1848, p. 662, and December 27th, p. 708.
[2] *Dub. Hosp. Gaz.*, 1845, 1, p. 109.
[3] *R.* v. *Pym*, Lancaster Ass., March, 1902.

A girl, *æt.* 19, took a strong decoction of the *leaves* to bring on the menses. The dose taken was a tumblerful for four successive mornings. Severe vomiting followed, and this was promoted by tepid water. Delirium came on, and the patient died eight hours after taking the last dose. It is stated that nothing of importance was revealed by an inspection of the body.[1] In another case a girl, *æt.* 13, took the leaves for a similar purpose. Death took place rapidly, without any other symptoms of poisoning than vomiting. On inspection there was congestion of the membranes of the brain, liver, and kidneys, a greenish colour of the contents of the stomach and intestines owing to the fragments of yew leaves, and stellated inflammation of the mucous membrane of the stomach and bowels.

A child, aged three years and a half, ate a quantity of *yew berries*. An hour afterwards the child appeared ill, but did not complain of any pain. It vomited part of its dinner, mixed with some of the berries. A medical man was sent for, but the child died in convulsions before he arrived. On inspection the stomach was found filled with mucus, and the half-digested pulp of the berries and seeds. There were patches of redness in the mucous membrane, and this was so much softened that it could be detached with the slightest friction. The small intestines were also inflamed.

A lunatic ate a quantity of the *berries*, and seven hours afterwards he was found dead sitting in a chair. On inspection of the body the right cavities of the heart were distended with fluid blood of a dirty plum colour. The mucous membrane of the stomach was reddened and softened with patches of black congestion. The duodenum was in a similar state. In the lower part of the small intestines there was a mass of the berries. The liver and other soft organs were much congested.[2]

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Poisoning by Tragopogon

The plant is common in many parts of England, and about Essex the seeds are known as gipsy nuts. Some years ago a boy in the London Hospital died from the effects of eating the seeds. The symptoms were those of an irritant at first, later they were of a narcotic type ; the boy died about the third day. The seeds were easily identifiable when parts of the plant were brought for inspection. The *post-mortem* appearances were negative except for one slight patch of inflammation in the stomach. For a full account of the case *vide* the London Hospital Register.

Poisoning by Veratrum album and viride

Source and Method of Occurrence. These plants, commonly known as white and green hellebores, are both of South European origin. They are quite distinct from the true hellebores. Their toxic principles have been investigated by Wright and Luff, who found veratrine and cevadine. Commercially " Veratrine " is the term used to designate a mixture of alkaloids extracted from the hellebores and sabadilla. It forms a grey non-crystalline powder, scarcely soluble in water even on boiling ; but is more readily dissolved by alcohol and ether. It has a slight alkaline reaction, and combines with acids, forming soluble salts. Protoveratrine is the most important active principle of white hellebore. It differs from cevadine in its effect on muscles and on the medulla. There are a number of other alkaloids which have not been properly investigated either chemically or pharmacologically.

[1] *Lancet*, 1870, 2, p. 471.
[2] *Med. Times and Gaz.*, 1870, 2, p. 446 ; see also 1871, 1, p. 386, for another fatal case

Toxicity and Fatal Dose. Although protoveratrine is highly toxic, vomiting occurs so briskly that it is rarely fatal. Death has been reported after 18 grains of white hellebore powder. A physician prescribed for one grain of veratrine divided into fifty pills, and three were directed to be taken for a dose. Not long after the first dose had been swallowed the patient was found insensible, the surface cold, the pulse failing, and there was every symptom of approaching dissolution. She remained some hours in a perilous condition, but ultimately recovered. Supposing the medicine to have been well mixed, and the pills equally divided, not more than one-sixteenth of a grain of veratrine was here taken. This case proves that the alkaloid is capable of exerting a powerful effect.

Symptoms. Veratrine has an aconitine-like effect on the central nervous system and a specific effect on muscle. There is first a burning tingling sensation in the mouth and throat, followed by numbness. This spreads to the surface of the body, and salivation and profuse sweating occur. Great oppression is felt in the epigastric region, together with abdominal pain, colic, vomiting and profound prostration. After a time there is fibrillary contraction in the muscles. Death occurs from respiratory failure. In a case reported by Blake, nearly three grains of veratrine were accidentally swallowed by an adult. The patient complained of giddiness, sickness, constriction of the throat, thirst, diarrhœa with tenesmus, and a tired, weak, faint feeling. The tongue was swollen, and the mouth and throat were sore; the pupils were extremely contracted, the respirations hurried, and the pulse was quick and small; micturition was frequent. A continued tingling was felt over the entire body, with occasional fits of itching in different parts. There was no sneezing. Recovery took place under treatment, the irritation of the skin being the last symptom to subside.

Treatment must be on general principles, as for aconite poisoning.

Post-mortem Appearances. There is nothing characteristic, but evidence of irritation of the mucous membrane of the stomach may be observed.

Analysis. Veratrine has a hot, acrid taste, without any bitterness. Applied to the mucous membrane of the nostrils it causes violent sneezing. Strong nitric acid gives to it a light red, turning to an ochreous, colour. Concentrated sulphuric acid, when heated with the powder, or a residue containing veratrine, produces an intense persistent crimson-red colour. With sulphuric acid in the cold, the veratrine alkaloids form a yellow solution which changes to orange and then red, and has a greenish-yellow fluorescence. (Salicin treated with sulphuric acid turns red immediately without heating; narcotine gives a similar reaction, but takes hours to acquire the red colour). Boiled with concentrated hydrochloric acid, veratrine yields a blood-red colour which is remarkably stable and persists for days or weeks.

If a little veratrine is mixed with five or six times its amount of cane-sugar, and moistened with concentrated sulphuric acid, a yellow colour is first produced, which changes to green and finally to blue. With ammonium selenate and sulphuric acid veratrine yields a brownish yellow, which changes to rose red. Veratrine dissolves in nitric acid to

a yellow solution. If this is evaporated and the residue is moistened with freshly prepared alcoholic potash a reddish-purple colour is produced (atropine and strychnine behave similarly).

If a small quantity of a solution of the drug is injected into the dorsal lymph-sac of a frog, the animal becomes clumsy in its movements owing to the fact that it cannot relax its muscles.

Cases. A gentleman swallowed experimentally one drachm of tincture of green hellebore (*Veratrum viride*), equal to twelve grains of the powder. He was found soon afterwards in a collapsed state, the features sunk, the skin cold and covered with a profuse clammy sweat, the pulse scarcely perceptible. He complained of intense pain in the region of the stomach. There was no purging. These symptoms were relieved by treatment, and the next morning the patient had recovered.[1]

A woman, aged sixty-six years, drank a decoction of fifty grammes of white hellebore in a litre of water. Symptoms appeared very quickly, and consisted of vomiting, impaired vision, rigidity of the limbs, weak pulse and oliguria. Camphorated oil and caffeine were injected and caused a rapid cure. Oliguria with bloody urine persisted for three days.[2]

Nivet and Giraud have published the history of a criminal application of powdered veratrum, administered in repeated doses in the food. Two brothers, aged twenty-one and twenty-three years respectively, died; the mother, who was very ill, recovered.

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Group 8.—POISONS OF ANIMAL ORIGIN

As a group, poisoning by foodstuffs should be inserted here, but it is preferable to deal with it separately. This exclusion leaves us only cantharides and snake venom to consider.

Poisoning by Cantharides

Source and Method of Occurrence. The substance is the dried body of a beetle (*Cantharis vesicatoria*), used in medicine externally as an irritant and (rarely) internally for its effect upon the kidneys. The active principle is known as Cantharidin. Cantharides have been administered not infrequently, either in the state of powder or tincture, for the criminal purpose of procuring abortion. Out of 1,620 fatal cases of poisoning in four years there were only two which were ascribed to cantharides.

Cantharides have been in some cases wantonly used, and with great danger to life, with a view to exciting sexual feelings. The doses in which it has been given have been such as to cause symptoms of irritant poisoning.

In November, 1859, six female servants in a gentleman's family, as well as the master and mistress, were attacked with the symptoms of poisoning by cantharides. It appeared that the coachman of the family had shortly before the occurrence

[1] *Med. Times and Gaz.*, 1863, 1, 5.
[2] Mahen, *Jour. Pharm. et de Chemie*, 1925, vol. 2, No. 5, p. 185.

purchased an ounce of this poison ; that he had put the cantharides into beer and coffee, and had thus poisoned the whole household. He was tried, but acquitted of any indictable offence, on the ground that his intent was not to murder. It was this case which led to an alteration in the law.

At the Liverpool Lent Assizes, 1861,[1] a man was indicted for administering powdered cantharides to a woman.

The accused had mixed it in a cup of tea ; the woman took a portion of the tea, and suffered from vomiting and other symptoms produced by this substance ; she skimmed a quantity of the powder from the tea, on which it floated, and its nature was then determined. The accused was convicted of the act of administration, but a question arose in reference to the intent. The jury found that he had administered the powder with the intent to excite the sexual passions of the woman, for which the new statute had not provided, as this makes the offence to depend only on the intent to injure, aggrieve, or annoy.

Tincture of cantharides has been deleted from the Pharmacopœia and there is now no official preparation for internal use.

Toxicity and Fatal Dose. Cantharidin is readily absorbed from all surfaces including the skin. The *quantity* required to produce serious symptoms, or to destroy life, has been a frequent subject of medico-legal inquiry. In a trial which took place at Aberdeen in 1825, it appeared that a drachm of the powder had been administered ; severe symptoms followed, but the person recovered. A witness said that he had given ten grains of the powder as a medicinal dose. In three cases, robust healthy negroes, a drachm of the powder, mixed with six ounces of rum, was taken by each person. They suffered severely, but recovered within about ten days. In these cases, irritation of the urinary organs did not appear until after the men had been bled.

The smallest quantity of the powder which has been known to cause death was in the case of a young woman, quoted by Orfila. The quantity taken was estimated at *twenty-four grains* in two doses. She died within four days ; but as abortion preceded death, this may have been concerned in accelerating that event. Her intellect was clear until the last. In one instance a man recovered after having taken *two drachms*.[2] An *ounce* of the tincture has been known to cause death. This dose was taken by a boy, *æt.* 17, and he died within fourteen days. This is, perhaps, the smallest dose of the tincture which has proved fatal. Four drachms and even six drachms have been taken ; and although the usual symptoms followed, the parties recovered. Six drachms of the tincture were administered to a girl, *æt.* 17. The questions here arose whether half an ounce was sufficient to kill a person, as also what proportion of cantharides was contained in an ounce of the tincture. One ounce of the tincture is equivalent to six grains of the powder ; but as the proportion of *cantharidin* is subject to variation, it is not unlikely that the tincture varies in strength. The powder cannot be readily administered, since a large portion of it floats for a time on any liquid with which it is mixed, and attracts attention by its peculiar appearance.

Symptoms. When taken in *powder*, in a dose of one or two drachms, it gives rise to the following symptoms : a burning sensation in the throat, great difficulty of swallowing, violent pain in the abdomen, nausea, and vomiting of bloody mucus ; there is also great thirst and dryness of the throat, or sometimes salivation. As the case proceeds a

[1] *R.* v. *Wilkins.*
[2] *Med. Gaz.*, vol. 42, p. 873.

dull heavy pain is commonly experienced in the loins, and there is an incessant desire to void urine, but only a small quantity of blood or bloody urine is passed at each effort. The abdominal pain becomes violent and griping. Purging may supervene; but this is a symptom which is not always observed: the matters discharged from the bowels are mixed with blood and mucus, and there is often tenesmus (straining). In the fæces, as well as in the vomited liquids, shining green or copper-coloured particles may commonly be seen on examination, whereby the poison, if it has been taken in powder, may be detected. After a time there is severe priapism, and both in the male and female the genital organs are swollen and inflamed. In one instance abortion was induced, probably owing to excitement of the uterus from the severe affection of the bladder, for there is no proof that this substance acts directly on the uterus to induce abortion. With respect to its aphrodisiac properties, these can seldom be excited in either sex, except when the substance is administered in a dose which would seriously endanger life. When the case proves fatal, death is usually preceded by faintness, giddiness, and convulsions.

When a tincture is taken symptoms are more speedily induced, and the burning sensation and constriction of the throat and stomach are more strongly marked. These symptoms are often so severe as to render it impossible for the person to swallow, and the act of swallowing gives rise to excruciating pain in the throat and abdomen.

Applied to the skin, cantharides causes redness, pain and vesication. It may be absorbed from the skin and cause general symptoms.

Treatment. Empty the stomach and treat on general principles. Fatty substances must be avoided, but demulcents and albumen may be given in quantity. Alkalies should be given, for the irritation of the urinary tract is thus lessened.

Post-mortem Appearances. In one well-marked instance of poisoning by this substance, the whole of the alimentary canal, from the mouth downwards, was inflamed. The mouth and tongue seemed to be deprived of their mucous membrane. The ureters, kidneys, and internal organs of generation were also inflamed. In another instance, in which an ounce of the tincture was swallowed, and death did not occur for fourteen days, the mucous membrane of the stomach was not inflamed; but it was pulpy and easily detached. The kidneys, however, were inflamed. The brain has been found congested, and ulceration of the bladder is said to have been met with. There are few fatal cases reported in which the appearances have been accurately noted. Indeed, the greater number of those who have taken this poison have recovered. In a case which occurred to Saunders, death took place within about twenty-four hours. The deceased must have taken the greater part of half an ounce of cantharides in powder. The symptoms were such as have been above described. On inspection the vessels of the brain were filled with dark-coloured blood, and the ventricles were distended with serum. Both lungs were highly engorged with dark-coloured blood. The gullet was partially inflamed, and there were patches of inflammation on the mucous coat of the stomach, which had become detached in several places. The same inflammatory appearance existed in the small intestines, in the folds

of which the powder of cantharides was abundantly present. The vessels were distended, and the liver was engorged with dark blood. The gall-bladder was much distended with bile, and none of this secretion appeared to have passed into the bowels. The spleen and kidneys were highly congested; the ureters were inflamed; the bladder was contracted and empty, and its internal surface pale. The glittering of the particles of cantharides on the viscera during the inspection by candlelight was very remarkable.[1] Cantharides powder has no local chemical action: the poison is a pure irritant, and the effects observed on the stomach are entirely due to irritation and inflammation.

Analysis. The wing-cases and other parts of the beetle must be carefully looked for. For the detection of the powder, the suspected liquids, mixed with alcohol, should be spread on sheets of glass, and allowed to evaporate spontaneously to dryness. The shining scales will then be seen, on examining by reflected light either one or both surfaces of the glass.[2] As the powder is insoluble in water, some portion of it may be obtained by washing and decantation. The sediment may be examined on a glass slide with the microscope.

Cantharidin is a neutral crystallisable principle which forms on an average only $\frac{1}{250}$th part of the beetles. For its detection acidify and digest the suspected solid or the liquid contents of the stomach (evaporated to a syrup) in successive quantities of ether, concentrate these ethereal solutions by slow evaporation, and then observe whether the concentrated liquid applied to the skin of the lips, or the lobe of the ear, produces blistering. The $\frac{1}{100}$th of a grain of cantharidin dissolved in ether is said to possess blistering properties.

Chloroform may be used for the separation of cantharidin from the tincture or from an alcoholic or aqueous extract of the contents of the stomach. An ounce of chloroform may be frequently shaken with the acidified suspected matters and left in contact with them twenty-four hours. The chloroform is then separated by a funnel, filtered, and allowed to evaporate spontaneously in a watch-glass. A pellet of lint of the size of half a pea, pulled out, is moistened with a drop of olive-oil, and the residue in the watch-glass taken up by it. This is placed upon the arm, lobe of the ear, or lip, and covered with goldbeaters' skin. When taken off in three or four hours, the skin is very red, and on wiping it with chloroform a vesicle may have been produced.[3] The quantity of cantharidin detected in this way has amounted to only the $\frac{1}{80}$th part of a grain. This mode of operating is preferable to the use of ether, as cantharidin is less soluble in ether than in chloroform. Half an ounce of the tincture of cantharides will yield to chloroform a crystallisable principle, having the characters assigned to cantharidin. In practice it will be found advisable to concentrate the liquid as much as possible before adding the chloroform.

Cantharidin crystallises in flat plates or in right-angled four-sided columns. It is soluble in alkalies, ether, benzene, or chloroform, but insoluble in water. It sublimes on heating, but gives no simple characteristic chemical reactions.

[1] *Med. Times*, February 3rd, 1849, p. 287.
[2] *Ann. d'Hyg.*, October, 1842.
[3] *Chem. News*, February 14th, 1863, p. 78.

The evidence of the presence of cantharides, or of their having been taken, is necessary to support a criminal charge; for however unambiguous the peculiar effects on the generative and urinary apparatus may appear to render the symptoms produced by this poison, the medical jurist should be aware that similar symptoms may proceed from disease. An important case of this kind has been reported.[1]

A young lady was suddenly seized with vomiting, thirst, pain in the loins, strangury, and considerable discharge of blood from the urethra; the generative organs were swollen and painful. She died within four days. She was governess in a family, and there was some suspicion that she had been poisoned with cantharides. The stomach and the kidneys were found inflamed, and the bladder also. This contained about two ounces of blood. No poison was detected; and indeed it was pretty certain, from the general evidence, that none could have been taken.

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Cases. At a meeting of the Royal Academy of Medicine in Ireland in May, 1904, Dr. F. C. Martley brought forward a case in which an elderly woman had been poisoned by cantharides. About an ounce of the powdered drug had been administered to her in a glass of rum, but in spite of the amount having been so large, she survived for thirty-one and a half hours.

Snake-poison and Snake-bite

Source and Method of Occurrence. The fact that about 20,000 people die from snake-bite in India every year has led to numerous investigations, and to-day we possess a considerable body of knowledge on the action of snake venom; and some progress has also been made with regard to its treatment. With the exception of New Zealand, Ireland and the Oceanic Islands, venomous snakes are found all over the world. Zoologists have divided them into two main groups, Colubridæ and Viperidæ.

In Europe small vipers or adders (*Viperus berus*, *V. aspis*, *V. ammodytes*) are found and occasionally cause death, especially in children. In India, China and Southern Asia extremely deadly snakes are found, notably the cobra (*Naja tfipudians*), the hamadryas (*Naja bungarus*), and the kraits (*Bungarus cæruleus* and *B. fasciatus*). The venomous vipers include *Daboia russellii* and *Echis carinata*. In America the chief venomous snakes are viperine and especially the sub-family Crotalinæ (rattlesnakes), among the most feared being *Crotalus terrificus*, *C. scutulatus*, *C. horridus*, the copper-head *Ancistrodon contortix* and the moccasin (*A. piscivorus*). In Africa *Naja haja*, *Cerastes cornutus* and *Echis carinata* are responsible for a number of deaths. In Australia the venomous snakes, though rarely large, are often very deadly, notably *Notechis sclitatus*, *N. pseudechis*, *Denisonia superba* and *Acanthophis antarticus*. There are many sea snakes with very deadly qualities.

[1] *Med. Gaz.*, vol. 12, p. 431.

The poison is secreted by the cells of a racemose gland, the ducts of which, one on each side of the head, open into the poison fang, and while this is inserted into the victim the venom is instilled into the wound at the same moment.

Toxicity and Fatal Dose. Snake venom is practically inert when swallowed into the stomach. To produce lethal effects it must be injected into a blood vessel or be absorbed by the tissue lymphatics. The modern study of the action of snake venom dates from the admirable work of Weir Mitchell and Reichert, followed by that of Calmette, Fraser, Lamb, Martin, Noguchi and many others. As a result of their researches it is now known that different venoms vary greatly in their toxicity and in their action. Venoms contain three main groups of poisons, viz.: (1) neurotoxins which act on the nerve cells or peripheral terminations; (2) cytolysins, acting on red blood corpuscles, endothelial cells, leucocytes, etc.; (3) fibrin ferments, causing intravascular clotting. In different snakes these toxic groups may be represented in different proportions, some being absent, and this no doubt accounts for the variations in symptoms produced in the victims.

Kellaway[1] considers that viperine venoms though strong in thrombins are weak in neurotoxins which produce paralysis of respiration. They are therefore less toxic than the venoms of the Colubridæ, which though weak in thrombin are strongly neurotoxic.

Duration. The action of the poison is remarkably rapid, death frequently taking place within a quarter of an hour and usually within a few hours.

Symptoms. Cobra Bite. In a patient bitten by a cobra the first sensation is a burning pain at the site of the bite, which becomes red and swollen. In perhaps half an hour the patient feels sleepy and weak in the legs and is soon unable to stand. Salivation, paralysis of the tongue, aphonia and aphagia rapidly supervene, and the paralysis becomes general. Breathing is slow and ultimately ceases.

Bite of Rattlesnake. The pain in the wound is very severe, and great prostration, cold sweats, nausea and vomiting with feeble pulse and dilated pupils may occur within fifteen minutes. Local hæmorrhagic extravasations occur and are very prone to suppurate, so that even if the patient survive the acute intoxication, he may succumb later to sepsis.

Bite of European Viper. The bite is followed by burning pain, and the affected part becomes swollen, œdematous and discoloured. Great prostration, accompanied by vomiting, nausea and diarrhœa, are common symptoms, and cardiac failure often supervenes.

Prognosis. The prognosis of snake-bite is difficult to estimate. Martin and Lamb state that the mortality, in India and South America, does not appear to be more than 30 per cent. of those bitten, and it may be very considerably less.

[1] *B.M.J.*, June 10th, 1933.

Post-mortem Appearances. The *post-mortem* appearances of death from snake-bite in the human subject have not been very extensively studied. The connective tissue in the neighbourhood of the bite is infiltrated with a sanguinolent fluid. The blood is usually liquid, and varying degrees of congestion are found in the parenchymatous organs, especially the kidney.

Treatment. In 1894 Calmette made the important discovery that animals repeatedly injected with increasing doses of snake venom yield sera which possess antitoxic properties and are thereby able to neutralise the toxic effects of snake venom. Fraser confirmed the work of Calmette. At first it was believed that anti-venene neutralises the effects of all kinds of venom, but Lamb especially has shown that this is not the case. The hopes that were raised that in anti-venene a sovereign therapeutic remedy for snake venom had been found have not been realised in practice. The acuteness of the poisoning is also a great difficulty in the treatment of those bitten. Of other important methods of treatment, measures should be taken as early as possible to prevent the poison from entering the system and to counteract its effects. The application of a ligature to the affected limb above the bitten area should be carried out at once, and as soon as medical aid is at hand free incision should be made into the region of the bite and the wound thoroughly washed out with a strong solution of permanganate of potash or crystals of permanganate rubbed into the incision.

Scorpion stings are also dangerous to life by reason of the hæmolytic poison which is injected. They cause local pain or numbness, muscular spasms and extreme restlessness, pyrexia, and allergic symptoms usually of an asthmatic type. Death occurs most frequently in young children; adults usually recover. *Treatment* is by sedatives to permit of rest and sleep, but it is not advisable to use morphine for this purpose. The use of specific anti-serums has given promising results.

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Vide also *Lancet*, 1904, 1, p. 349, for an article by Dr. Leonard Rogers, on the action and treatment of the poison. A valuable table is there given.

Group 9.—POISONING BY FOODSTUFFS

Source and Group of Occurrence. In all cases of poisoning by foodstuffs the symptoms are caused by one or other or both of two things: (1) a living microscopic organism; (2) chemical poison of greater or less virulence. There are, however, several distinct methods by which foods may become poisonous, and it is exceedingly important to bear them in mind.

1. The food may not be fit for human consumption originally; *e.g.*, certain molluscs, fungi, fish, etc.

2. The food itself may be perfectly wholesome in every way, but it may disagree with its host from (*a*) unsuitability to circumstances as to age, exercise, illness, etc.; (*b*) idiosyncrasy.

3. The food may contain definite poison owing to the animal having

fed on plants poisonous to human beings (*e.g.*, belladonna), or it may contain definite metallic poisons.

4. It may contain the germs of specific diseases ; *e.g.*, typhoid fever in oysters, scarlet fever or diphtheria in milk.

5. It may contain other pathogenic bacteria or their toxins.

6. It may contain simple non-specific chemical products of decomposition (" ptomaines ").

7. It may contain parasitic worms in some stage of development.

8. It may be injurious to man owing to added preservatives.

1. **Food Originally Unfit.** Particulars under this head need hardly be given. Certain kinds of fish, such as the tetradons, including the Japanese fugu, distributed widely in Eastern waters, and certain European fish, such as the barbed sturgeon and carp, are alleged to be poisonous at certain periods. The roe appears to be the most toxic part of many fish. The subject of mushroom poisoning has already been dealt with on pp. 704 *et seq.*

2. (*a*) **Unsuitability.** It is unnecessary to do more than mention this.

Indigestion and acute gastritis in adults are illustrations belonging to clinical medicine, but cases of this nature occasionally require some investigation before poisoning, malicious or otherwise, can be excluded.

(*b*) **Idiosyncrasy.** Certain kinds of food are found to produce symptoms of poisoning occasionally. In some instances this poisonous effect appears to be due to idiosyncrasy, for only one person out of several may be affected. These cases are of importance to the medical jurist, since they may give rise to unfounded charges of criminal poisoning. In the absence of any demonstrable poison, we must test the question of idiosyncrasy by observing whether more than one person is affected, and whether the same kind of food given to animals produces symptoms of poisoning. If, with this latter condition, several persons are affected simultaneously, we cannot refer the effects to idiosyncrasy ; they are most probably due to the presence of a poison. The symptoms which occur in these cases are broadly of three kinds : gastro-intestinal irritation ; skin irritation, in the form of urticaria or eczema ; asthma and other pulmonary symptoms. Œdema is also a common feature. The phenomena appear to be anaphylactic in origin. Among articles of food which have caused symptoms of irritant poisoning **shell-fish** easily hold the first place, and of all the varieties of shell-fish none have so frequently given rise to accidents as the common mussel, though crabs and lobsters are also very frequently responsible for cases seen in hospital casualty rooms (see also " Arsenic Poisoning "). The symptoms produced are uneasiness and sense of weight in the stomach ; sensation of numbness in the extremities ; heat, dryness, and constriction in the mouth and throat ; shivering ; difficulty of breathing ; cramps in the legs ; swelling and inflammation of the eyelids and face ; a profuse secretion of tears ; and heat and itching of the skin, followed by an eruption known as urticaria from its resemblance to the swellings caused by the sting of a nettle. These symptoms are sometimes accompanied by colic, vomiting,

and purging. They may occur within ten minutes or a quarter of an hour; but their appearance has been delayed for twenty-four hours. There is generally great exhaustion and debility.

"A man ate about twenty mussels. He soon began to have nausea and griping pains. In half an hour he vomited and was purged several times. He then felt faint and dizzy. When seen two hours and a half after eating the fish he was collapsed, and the pulse was almost imperceptible, though not much increased in frequency. The pupils were natural. There was loud wheezing and rattling respiration, interrupted by frequent yawnings and sighings. Notwithstanding free stimulation with brandy, the patient was continually fainting; and he had frequent attacks of clonic spasms of all the muscles of the body. He was calm and conscious, had no pain, but complained of great thirst. There was itching of the skin, and an erythematous rash on the chest. Under the administration of opium and ether this condition speedily passed off." [1]

The symptoms however, are not invariably those of irritant poisoning.

A man who ate two or three quarts of mussels scraped off a vessel was absolutely unconscious when admitted into hospital. His face was livid, the pulse almost imperceptible, and he breathed only once or twice per minute by convulsive gasps. The pupils were widely dilated, and the reflexes abolished. Clearly he was asphyxiated, and he died about ten hours after being discovered in an unconscious state. Two other men suffered to a lesser degree at the same time.[2]

Segers described a chronic form of mussel-poisoning among the inhabitants of Tierra del Fuego, who ate mussels, eleven to twenty-two pounds daily, almost to the exclusion of any other kind of food. The symptoms are marked icteric discoloration of the skin, enlargement and subsequent atrophy of the liver, and hæmorrhages from the mucous surfaces ushering in death. When the mussels are in good condition they are seldom injurious; but when they are thin they are often poisonous. Segers attributed this to the death of large numbers of mussels, their decomposition, the consequent formation of poisonous substances, and the absorption of these by the living mussels.[3]

The poisonous action of mussels can be referred neither to putrefaction nor to disease, nor in all cases to idiosyncrasy, since in one instance those mussels only which had been taken from a particular spot were poisonous; all persons who partook of them suffered, and a dog to which some of them were given was killed. Brieger has obtained from poisonous mussels a poisonous ptomaine, *mytilotoxine*, to which he has assigned the definite formula $C_6H_{15}NO_2$, and to which he attributes the toxic effects of mussels ("Die Ptomaine").

A fatal case came under Sir Thomas Stevenson's notice in 1895, and another was the subject of an inquest at Bolton, in February, 1904.

It is probable that in some at least of the above cases some of the other factors mentioned above came into play. Of other well-known foods which some people cannot touch, **strawberries, cheese,** and **eggs** are the best examples, but more extensive observation has shown that almost any protein may cause symptoms of anaphylaxis in some individuals.

Linseed can hardly be called a food, but for all that it is a food for domestic animals, and certainly one would have no dread of it if eaten,

[1] *B.M.J.*, 1882, 1, p. 939.
[2] *Lancet*, 1888, 2, 568.
[3] *Prensa* of Buenos Ayres, July 23rd and August 1st, 1891. *B.M.J.*, 1891, 2, Sup., p. 169.

so that the following case is well worth recording as one of peculiar idiosyncrasy. It is published in the *Lancet*, 1903, 2, p. 1428, by Dr. Hollick, of Knowle :—

" The patient, a robust man, consulted me on May 19th, 1903, for an inflamed and protruding internal hæmorrhoid which had become strangulated by the external sphincter, and was giving him very severe pain. I ordered him to bed, and as he had already applied anodyne ointments and lotions without obtaining relief, I also directed a hot linseed-meal poultice to be applied to the inflamed and painful hæmorrhoid. This was done at about 3 p.m., and within four hours afterwards the patient experienced the following symptoms, which I have recorded in his own words :—

" ' To commence with, I experienced a peculiar sensation in the throat and mouth, as though they were lined with velvet, the throat contracting considerably ; there was a scaly feeling in the skin of the hands and feet. The skin all over my body, but more particularly on the thighs and legs, went " anserine," having the appearance as though a small bladder of water surrounded each hair at its root ; the skin changed to a colour between that of red and purple ; after this the irregularities on the skin became more pronounced. My face was discoloured, and there was a rush of blood to my head. This discoloration of skin was accompanied by irritation. My hearing was affected. I went very deaf, and I was also unable to see well. My heart was beating very forcibly and rapidly, and I had a strong hysterical feeling. I counted my pulse, and timed it to be 120 per minute. I felt on the border of delirium. My chest seemed restricted, particularly at the lower part. These symptoms lasted about three-quarters of an hour, and on diarrhœa supervening they were very much mitigated. I then had an attack of vomiting, the vomit being of a dark colour, although I had taken nothing but milk foods for the past forty-eight hours. After this the symptoms gradually passed off, the velvety feeling at the throat and mouth being the last to disappear. On two previous occasions I had been " poisoned " by linseed. On the first occasion I was walking through a field in the east of England where labourers were engaged in the operation of stacking the linseed, and I ate a few of the seeds. Within three or four hours afterwards I experienced symptoms similar to those narrated above, only to a less degree. I ate about twelve seeds only, and the duration of the symptoms was from two to three hours. About two years afterwards I ate two lozenges of " linseed and liquorice," and the same symptoms were again experienced.'

" The patient sent for me at 8.30 p.m., and, as he resided some distance out in the country, I did not see him until 9 p.m.—*i.e.*, two hours after the commencement of the symptoms. He was then in a state of collapse, with quick and feeble pulse, a feeling of nausea, and with cyanosed condition of the face and extremities. His respiration was quiet, and the diarrhœa had ceased, but he had a marked condition of cutis anserina. He lay curled up on his side in bed, and was very prostrate ; he at once told me that he had been poisoned by linseed, as recognised from his previous experiences. He was given an ounce of brandy in hot water, and hot bottles were placed to his feet and body ; he passed a good night, and on the next day was in normal health again.

3. **Poison in the Food, viz. a Metallic Irritant or from the Plants on which the Animal fed.** The milk and cheese of some of the North American provinces is said to be occasionally rendered poisonous by the fact that cows pasture at certain seasons on vegetables of a noxious kind. In 1865, twelve cases of poisoning from this cause were reported. The symptoms came on in about three hours after cheese had been eaten. There was severe pain in the stomach, cramp, violent vomiting of a greenish fluid, soreness of the throat, and a cold clammy condition of the skin. All recovered, recovery being preceded by profuse perspiration.[1]

Vaughan attributes the poisonous character of decomposed cheese to a ptomaine, *tyrotoxicon* (diazobenzene butyrate), which has been isolated from poisonous cheese and milk.

As pork is sometimes salted in leaden vessels, lead may be found in it.

[1] *Edin. Med. Jour.*, 1865, 1, 854.

Poisoned game is occasionally sold. The game may be quite free from putrefaction, but noxious from the poisoned grain which may have caused death. It is a common practice to steep grain in a solution of arsenic previous to sowing, and pheasants, partridges, and other birds may be accidentally destroyed by eating the grain. In some instances, grouse and other game are maliciously destroyed by the laying of corn steeped with arsenic, strychnine, or other poisons, in the localities where the birds abound. There is no law, except the Sale of Food and Drugs Act, to prevent the sale of poisoned game by poulterers, and there is no precaution which can be taken by the purchasers except by observing whether the birds have or have not been shot.[1]

The **greening of vegetables** by copper is another illustration. Mutton is occasionally poisoned by mercury. Tin and other metals may contaminate tinned foods. Sprays used for fruit pests may cause symptoms of poisoning, as, for example, arsenic on apples.

For an illustration of poisoning by **honey** gathered from poisonous plants, *vide B.M.J.*, 1899, 2, p. 674.

4. **Disease arising from Specific Microbes in the Food.** Of this class of illness from food, examples are only too common. Outbreaks of enteric fever have upon several occasions been traced to oysters and also to polluted water and milk.

In a similar manner scarlet fever and diphtheria have been upon many occasions traced to milk supplies.

This part of the subject, however, belongs too much to public health to warrant more than a simple mention here ; and the reader is referred for further details to " Food Poisoning and Food Infections " (Savage, 1920), and " Food Poisoning : a Study of One Hundred Recent Outbreaks " (Savage and Bruce White) ; Med. Res. Council Report No. 92, 1925 ; also Report 91, on Salmonella group of Bacteria.

5. **Presence of Pathogenic Organisms and (or) their Toxins.** *The Salmonella Group.* It has been shown that, in the majority of outbreaks of food poisoning which have been properly investigated, the cause has been traced to the presence of bacteria of the Coli enteritis group or their toxins. This group is known as the Salmonella group, and includes the organisms of paratyphoid fever. The members of the group which are most commonly responsible for outbreaks of food poisoning are *B. Aertrycke* and *B. enteridiidis*, of Gaertner, but many others have been incriminated with considerable frequency.

These organisms are readily killed by heating to 60° C. for half an hour, so that it is only rarely that an outbreak occurs from well-cooked food that has not been reinfected. Their toxins, however, are very resistant to heat, and sufficient toxin may be present to cause acute symptoms. Although all the bacteria are killed, symptoms due to the presence of toxins alone are recognised by the rapidity of onset after taking the suspected food, and also as a rule by the rapid remission of symptoms. Food may be sufficiently infected to cause toxic symptoms without any visible alteration in taste or smell.

[1] See on this subject " On Poisons," *Med. Gaz.*, vol. 42, p. 103.

The incubation period in salmonella poisoning has varied in different outbreaks from half an hour to forty hours, but the average is from six to twelve hours.

The symptoms consist of vomiting, abdominal pain and diarrhœa. These cardinal symptoms may be accompanied by prostration, collapse and sometimes rigors. There is usually some fever. Less common symptoms are headache, giddiness, numbness and cramp in the limbs.

Staphylococci, Streptococci and other Organisms. Within comparatively recent years, it has become apparent that some strains, fortunately uncommon, of staphylococci and streptococci may produce toxins which are responsible for a severe form of food poisoning. As the illness appears to be due as a rule to the presence of preformed toxins, the incubation period is short—usually only a few hours. The clinical features are nausea, vomiting, diarrhœa and prostration. Similarly, it would appear that certain rare strains of the widespread *B. proteus* may produce toxins which are capable of causing food poisoning.

Botulism (Allantiasis). This is a form of poisoning with clinical symptoms which differentiate it from the ordinary types of meat poisoning associated with *B. enteritidis* and its congeners. Botulismus has been caused by consumption of sausages (especially those containing blood and liver), meat, salted, or smoked and fish (Ichthyosismus). The symptoms are almost exclusively referable to the central nervous system, and include motor paralysis, especially in connection with the cranial nerves, mydriasis, ptosis, diplopia, complete fixation of the bulbus, dryness of the mouth, aphonia, dysphagia ending in complete inability to swallow, dysuria, anuria and usually constipation. The cause of this remarkable state of affairs was discovered by van Ermengem in 1895 in an epidemic at Henegau and proved to be an obligate anaerobic bacillus (*B. botulinus*), which, like tetanus bacillus, is capable of producing a genuine toxin, which can cause all the symptoms of the disease. In comparison with the ordinary gastro-intestinal types of meat poisoning botulismus is a rare disease, but where found seems to be constantly due to van Ermengem's bacillus. A potent antitoxin has been prepared against the toxin of this microbe.[1] Botulism is fortunately uncommon in Britain. In 1922, at Loch Maree, Scotland, eight people were affected after eating sandwiches prepared with wild duck paste. All died within a week. In 1935, four fatal cases occurred in London.

6. "**Ptomaine**" **Poisoning.** Ptomaines are non-specific substances produced by the decomposition of animal proteins by various groups of putrefactive bacteria.

The investigations of Panum, Bergmann, Schmiedeberg, Selmi, Nencki, and especially of Brieger, have acquainted us with the symptoms which appear after inoculation with the soluble products of putrefactive bacteria. Such inoculation causes symptoms of convulsions and paralysis, and may prove fatal with symptoms of respiratory paralysis. It is distinguished from infection by the rapidity of onset after inoculation, by the fact that it occurs the more quickly and violently the larger the quantity of inoculated material, and, finally, by the fact that the disease

[1] Dickson, "Botulism," N.Y., 1918. See also *Analyst*, 1923, 48, 118-120, with reference to the outbreak of botulism in Loch Maree, Scotland.

cannot be transmitted further by inoculation with parts of the bodies of animals which have died in consequence of intoxication.

With regard to the nature of the degradation products of protoplasm formed during putrefaction, the work of Brieger showed that certain crystalline bodies could be isolated which, in accord with Selmi, he called ptomaines. To this group belong muscarine, choline, cadaverine, putrescine, neurine, neuridine, saprine, etc.

The formation of amines identical with the ptomaines of Brieger has been shown to occur in the normal human intestine. We may say that as far as our knowledge goes at the present time, it would appear to be established that certain poisonous amines may be produced by putrefactive processes similar to those formed in the normal intestine. Further, though such amines may be proved to be poisonous when injected, there is nothing to show that they are capable of causing ill effects when ingested in the ordinary way. If enormous quantities of putrid food passed into the intestines it is possible that the normal protective processes might be overcome, and toxic symptoms would then arise. It has to be remembered, however, that these bodies are produced in quantity only when putrefaction has advanced to a considerable extent, and in such cases the food would be obviously unfit to eat.

It may therefore be accepted that ptomaines are not the cause of food poisoning.

7. **Presence of Worms.** *Trichinosis.* The fatal malady arising from the introduction of the *Trichina spiralis* into the human body has attracted much attention. Keller has published some important facts illustrating the symptoms produced, and the mode in which this parasite causes death. He considers that it is a question well worthy of the attention of medical jurists whether many cases of death from suspected irritant poisoning, in which no poisonous matter could be detected in the body, may not really have been due to trichina disease.

The symptoms produced by the use of infected food are, in the first stage, those of intestinal irritation, loss of appetite, sickness, pain, general weakness of the limbs, diarrhœa, swelling of the eyelids and of the joints, profuse clammy perspiration, and wasting fever. Death is either the result of paralysis (from destruction of the muscular fibres) or of peritonitis and irritative fever. During the perforation of the coat of the intestines by these worms the mucous membrane becomes irritated and inflamed; pus is formed on its surface, and bloody evacuations are sometimes passed.

The noxious effects of this food on human beings are well illustrated by a series of cases which occurred at Hettstadt, in the Hartz mountains, in 1863.[1]

One hundred and three persons partook of smoked sausages made from a pig affected with trichinous disease. The sausages were fried, and served for dinner in the usual way. On the following day several persons who had partaken of this food were attacked with severe pain in the bowels, purging, loss of appetite, great prostration of strength, and fever. The number of persons attacked rapidly increased; symptoms of peritonitis and pneumonia appeared, and these were followed by paralysis of the intercostal muscles, and of the muscles in front of the neck. Eighty-three persons died from the effects of this noxious food, and the remainder were seriously injured in health. The remnants of sausage and of pork

[1] *B.M.J.*, 1864, 1, p. 75.

not eaten at the festival were examined, and were found to be swarming with encysted trichinæ.[1]

This parasitic disease does not apparently attack sheep, oxen, or horses, and beef is the safest of all descriptions of food, as no parasites have ever been discovered in it. They have not been found in the blood of animals whose muscles are liable to their attacks.

In suspected cases, if any of the food can be obtained, this must be examined for the parasite by the aid of the microscope. If the case proves fatal, the voluntary muscles of the deceased must undergo a similar examination. For further information the reader is referred to works on medicine and also on public health, the subject appertaining more to those branches than to forensic medicine.

8. **Excess of Preservatives.** Practically any poison may be added to food, either criminally or by accident, as has been described in the section dealing with poisons, but there remains the question of the addition of preservatives and colouring matters to food. This subject is of great importance, because the importation of food from abroad has become a national necessity, and in many cases the addition of a preservative is practically essential.

The preservatives principally used are boric acid and its compounds, salicylic acid and salicylates, sulphurous acid and sulphites, benzoic acid and benzoates, hydrogen peroxide and formaldehyde.

Any one of these substances may have a deleterious effect on the body by long-continued use, and in connection therewith the report of the Departmental Committee on Preservatives and Colouring Matters, 1909, should be consulted, also Savage, "Food Poisoning," 1920.

Under the Public Health (Preservatives, etc., in Food) Amendment Regulations, 1926, the manufacture and sale of food containing preservatives or colouring matters apart from those allowed by the regulations are forbidden. The regulations contain a schedule of articles of food which may contain such preservatives as sulphur dioxide or benzoic acid and the percentage allowed in each case. These regulations clear up the position of the preservation of food, limit the number of articles which may be preserved, and entirely prohibit all preservatives except those mentioned.

Analysis. In cases of suspected food poisoning, if any question is raised regarding a death taking place in such circumstances, the analyst's work, to be complete, must be very laborious. In the first place, fresh material must be at once submitted to a competent bacteriologist for examination, isolation and identification of the pathogenic bacteria. The result of this investigation may clear up the cause of death. If not, the analyst may be required to test for all metallic poisons (*vide* previous pages). He must next go through the complete process for the extraction of alkaloids (p. 265), and submit his results to a series of tabular experiments. Since ptomaines present certain of the properties of the vegetable alkaloids the possible presence of these bodies has to be considered.

Amongst the ptomaines which have been isolated are *putrescine*, *cadaverine*, *neurine*, *collidine*, *mydaline*, *tetanine*, *mytilotoxine*, and a host of others, some poisonous, others inert. L. Brieger[2] has described the largest number of ptomaines. He finds that Stas's process for their

[1] Casper's *Vierteljahrsschr.*, 1864, p. 286.
[2] "Die Ptomaine."

extraction from organic mixtures is not applicable to the larger number of ptomaines, and recommends their precipitation by aqueous and alcoholic solutions of mercuric chloride.

The formation of ptomaines being universal in corpses when undergoing slow decomposition, it might be presumed that they would be frequently observed in the bodies of persons who had died from acute arsenical poisoning. Selmi has succeeded in demonstrating that in these circumstances peculiar arsenical poisonous bases (arsines) are formed. In 1878 he reported two cases in which poisonous crystalline ptomaines were found in exhumed bodies, containing arsenic. The first subject was the body of a person exhumed fourteen days after burial, in a good state of preservation, containing much arsenic. In the search for alkaloids a small quantity of an alkaline substance, having a sharp bitter taste, was found. It reacted generally as an alkaloid. It afforded several colour reactions ; but the amount of material did not suffice for a complete chemical and physiological examination. Shortly afterwards Selmi obtained larger quantities of a ptomaine from an arsenical corpse exhumed a month after death. This base had likewise a sharp bitter taste. Its chemical reactions differed somewhat from those of the previously described alkaloid. It was highly poisonous when administered to a frog.

Though in these two ptomaines the presence of arsenic was not proved, Selmi afterwards discovered organic arsenical bases (arsines) in the stomach of a pig which had been preserved in a solution of arsenic. The tissues were not destroyed, and there was no putrescent odour perceptible. The liquid yielded, on distillation, an alkaline distillate which yielded crystals with hydrochloric acid. These, when moistened with caustic soda, exhaled an odour somewhat resembling trimethylamine. The presence of arsenic was ascertained in the hydrochloride of this volatile base, which yielded some alkaloidal reactions. Experiments made with twenty-four milligrammes (0·36 grain) of the substance showed it to be highly poisonous, and that it resembled strychnine in its physiological action. From the solid matter a volatile alkaloid was further extracted, but its small quantity prevented an accurate examination being made. From the residue of the distillation of the ether used in extracting these bases a third and non-volatile base was obtained, having a cadaverous odour. Its hydrochloric solution had an offensive odour and a bitter taste. It caused tingling when placed on the tongue, followed by loss of sensibility. The base yielded alkaloidal reactions, and contained arsenic. It was poisonous to frogs, but its action differed from that of the volatile arsines, and was somewhat similar to that observed in the ordinary action of the poisonous ptomaines. Torpor, paralysis, and stoppage of the heart in systole were the most prominent symptoms. The alleged existence of arsenical ptomaines is highly important, not only to the medical witness, but as affording a possible explanation of chronic arsenical poisoning produced by arsenical wall-papers, if Selmi is correct in his assertion that a volatile arsine possessing a highly poisonous action differing from that of arsenious acid is produced by the contact of arsenious acid and albuminous substances. Husemann thinks it likely that a similar product may be formed from the size employed in affixing the arsenical paper to a room, the moisture of the air playing a part in the formation of the arsine.

Selmi's researches may, as Husemann thinks, throw light upon an obscure page in the history of toxicology. It is asserted that the poisoners of the seventeenth and eighteenth centuries, Tofana and other professionals, understood how to make arsenic more potent. In Italy, the *acquetta di Perugia* was, according to tradition, a secret compound prepared by rubbing white arsenic into the flesh of a pig, and collecting the liquid which dropped from the flesh. It is possible that the activity of the arsenic was increased both by the formation of readily absorbable compounds of arsenic with the inorganic alkalies and by the formation of arsenical bases. The same object may have been in view in preparing *aqua Tofana*, with the addition, as is known, of the juice of the ivy-leaved toad flax (*Linaria cymbalaria*). Selmi and Vella are of opinion that in the *acquetta di Perugia* the concealment of the action of the arsenic on the one hand, and also of the tetanising poisons on the other, was accomplished; but this opinion, which is based upon an observation of Vella in a case of poisoning with arsenic and strychnine, does not accord with observations made on warm-blooded animals with a mixture of arsenite of potassium and strychnine, whereby the tetanising action of strychnine was not prevented, provided the alkaloid was given in poisonous doses.

Group 10.—MISCELLANEOUS POISONS

Poisoning by Mechanical Irritants

Various substances which act mechanically may, when introduced into the alimentary canal, result in death, *e.g.*, powdered glass, diamond dust, and dried sponge, or sponge soaked in grease.[1]

We have been unable to find any recent case of the kind in Great Britain, nor have we been able to find the case referred to by the *Lancet*, but the paragraph is worth reproduction as tending to elucidate the present position :—

" We understand that a case is shortly to come before one of the criminal courts in America which will be of unusual interest. A woman is charged with the murder of an aged husband by feeding him with glass ground up in an ordinary coffee mill. The glass is thought to have been given in oatmeal porridge. This method of poisoning is supposed to have been a favourite one in the sixteenth century, and is said to be still practised amongst savage tribes who have access to the needful material. Medical literature, however, contains but few recorded cases. Glass may be a ' noxious substance ' by virtue of the *mechanical* injuries which it can produce, but is not therefore a poison in the legal sense of the word. A case of this nature with references to previous observers is recorded in the *Edinburgh Medical and Surgical Journal* for 1824, p. 225. Dr. W. Turner, of Spanish Town, Jamaica, there relates that an attempt was made by a negro woman to poison a whole family with pounded glass. The persons on whom the attempt was made were seven in number, and none of them suffered any inconvenience. In the *Midland Medical and Surgical Reporter*, 1828, p. 47, Mr. William Hebb, surgeon, of Worcester, records in considerable detail the case of an infant who was destroyed ' by some person or persons administering to it a quantity of roughly pounded glass.' A considerable quantity of gritty powder, proved to be powdered glass, was found in the stomach, which ' was lined with a thick layer of tenacious mucus which was streaked with blood, and it required to be peeled off before the villous coat beneath could be exposed to view. This last was in a state of amazing vascularity.' Another case is reported in the *Allgemeine Wiener medicinische Zeitung*, 1863, 8, 244. A case of attempted suicide by this means is recorded in the *Boston Medical and Surgical Journal*, 1871, p. 191. A young girl, aged sixteen

[1] *Lancet*, 1892, 2, p. 1309.

years, desiring to end her life, pounded up a small glass bottle into fragments of the size of a split pea and under. Of these she swallowed a teaspoonful, taken at several times in bread. Although she experienced considerable pain and discomfort, she subsequently recovered. The favourable result may have been due to the bread protecting the stomach and intestines from injury. The small number of cases on record and the immunity which experimenters have enjoyed from harm after swallowing powdered glass would show that death produced by these means is not nearly as common as the remarks usually to be found in medico-legal text-books would lead the reader to imagine. In an inaugural dissertation published in Paris in 1830 by Le Sauvage, it is stated that two and a half drachms were given to a cat without injury, also that a dog took six or seven ounces in eight days without any symptoms manifesting themselves. Le Sauvage himself swallowed a considerable number of similar particles without sustaining any inconvenience. Professors Baudelocque and Chaussier in 1808 reported a case at Paris in which the prisoner was supposed to have poisoned his wife with pounded glass. This substance was actually found in the stomach of the deceased, and this organ as well as the intestines exhibited signs of great irritation. After a careful consideration of all that had been written on the subject, however, they gave their opinion that pounded glass is not a poison, and suggested that the glass in the stomach was derived from some vessel of that material being broken by her teeth during the convulsions which preceded death." [1]

Fine particles of glass occasionally pass into the intestinal canal from the fracture of small bubbles of glass in the inside of glass containers. It is possible that these may occasionally cause gastro-intestinal irritation.

We have seen several cases where powdered glass has been used for homicidal purposes in the East, but have not observed any serious effects from its use.

Poisoning by Proprietary Remedies and Quack Medicines

The following lists of patent medicines, etc., with their principal poisonous ingredients, though very imperfect, are not without interest, even though the sale and use of such preparations is much less common than previously :—

Battle's Vermin Killer. Strychnine, arsenic, and phosphorus.
Burnett's Fluid. Zinc chloride.
Correctives for Women. Aloes, ergot, pennyroyal, iron and arsenic, manganese dioxide, etc.
Fly Papers. Arsenic, also in wall-papers, and occasionally in the highly coloured papers in which sweets are wrapped (a most pernicious practice).
Godfrey's Cordial. Morphia.
Hair Washes and Face-paints. Mercury, arsenic, lead and bismuth, or chlorine, or hydrogen peroxide. Henna, phenylene diamines.
Headache Remedies sold by Chemists as proprietary. Phenacetin, aspirin, antipyrin, antifebrin, etc.
Mother's Friend.
Mrs. Winslow's Soothing Syrup. } Morphia.
Oil of Bitter Almonds. Hydrocyanic acid.
Paregoric. Opium.
Pigments used by Painters, etc. Chiefly lead, and zinc.
Powders for Dusting Infants. Occasionally contain arsenic with starch, fuller's earth, boracic acid.
Proprietary Disinfectants. Phenols and other coal-tar derivatives.
Rat Paste. Phosphorus, strychnine, arsenic, barium.

[1] *Lancet*, 1899, 1, p. 174.

St. Jacob's Oil. Aconite.
Seigel's Preparations. Aloes and saline aperients.
Sorrel. Oxalic acid.
Teething Powders. Calomel and opium.

Aperient and Liver Pills

Beecham's Pills. Aloes, ginger, and soap.
Baillie's Pills. Aloes, colocynth, oil of cloves, and soap.
Bile Beans. Cascara, rhubarb, liquorice and ol. menth. pip., coated with gelatine.
Cockle's Pills., *Barclay's Pills.* } Aloes, colocynth, and rhubarb.
Carter's Little Liver Pills. Podophyllin (gr. $\frac{1}{8}$) and aloes soc. (gr. $\frac{1}{8}$) in each pill.
Dixon's Pills. Taraxacum, podophyllin, jalap, and soap.
Holloway's Pills. Aloes, rhubarb, saffron, Glauber's salts, and pepper.
Page Woodcock's Wind Pills. Aloes, ol. carui, and soap.
Scott's Pills. Aloin and cascara, with a soap basis.
Whelpton's Pills. Rhubarb, aloes, ginger, pulv. ipecac., and soap.

Cough Mixtures and Lozenges

Keating's Cough Lozenges. Ipecacuanha, lactucaria, squill, liquorice, tragacanth, and sugar.
Owbridge's Lung Tonic. Balsam of tolu, oil of aniseed, and oil of cloves.
Balsam of Aniseed. Contains aniseed and other ingredients with gr. $\frac{1}{10}$ morphine in every ounce.

Preparations for Gout and Rheumatism

Blair's Gout Pills. The active ingredient is colchicum.
Eade's Pills. Sodium salicylate, guaiacum, and aloes.
Gloria Tonic. Colchicum, guaiacum resin, and sodium iodide.

Preparations for Headaches and Neuralgia

Antikamnia. Sodium bicarbonate, antifebrin, and (?) caffeine.
Bromidia. Potassium bromide, chloral, hyoscyamus, cannabis indica, oil of aniseed, syrup, and water.
Bunter's Nervine. Creosote, chloroform, camphor, balsam of tolu, and alcohol.
Kaputine. Antifebrin and sugar (coloured).
Kay's Tic Pills. Iron sulphate, quinine, and soap.

Preparations for Asthma

Crevoisier's. Belladonna, foxglove, stramonium, sage, and potassium nitrate in equal parts.
Hair's Cure. Potassium iodide and tar water.
Plant's Cigarettes. Leaves of stramonium, lobelia, and green tea.
Tucker's Cure. Atropine, cocaine, hyponitrous acid, and various balsamic extracts, administered by means of an atomiser.

Remedies for Obesity

Antipon, Silf, etc. Containing thyroid extract.
Dekrysil. 2·4 dinitro cresol.
Mrs. Frost's Anti-Obesity Remedy. The active ingredient is extract of fucus vesiculosus.

Grey's Specific. Contains 47·2 per cent. of free sulphur and a bitter (? gentian).

Russel's Anti-Corpulent Cure. Citric acid (twenty grains to half an ounce), glycerine, and water. The pink tablet contains the saccharine.

Trilene Tablets. Sugar and a vegetable constituent of unknown nature.

Ointments and Liniments

Cuticura. Vaseline and oil of bergamot.

Elliman's Embrocation. Acetic acid, turpentine, and white of egg.

Holloway's Ointment. Turpentine, resin, olive oil, lard, wax, and spermaceti.

Homocea. Camphor, oil of cajuput, and lard.

St. Jacob's Oil. Oil of turpentine, ol. succini, soft soap, and capsicum.

Lineel Liniment. Zinc and magnesium chlorides with glycerine.

Singleton's Golden Ointment. An imitation of ung. hyd. nit. dil.

Miscellaneous Preparations

Buer's Piles Cure. Ointment of galls, and hamamelis with lanoline basis; the powder contains precipitated sulphur, and magnesium carbonate.

California Syrup of Figs. Senna (active constituent), syrup of figs, and cinnamon.

Doan's (Backache) *Pills.* (1) White-coated aperient (dinner pills), podophyllin, aloin, rhubarb, and peppermint; (2) brown-coated (*backache*), oil of juniper and a resinous constituent (? benzoin).

Guy's Tonic. Phosphoric acid, tinct. cocci, infusion of gentian, and chloroform water.

Dalby's Carminative. Pulv. rhei, magnes. carb., glycerine, sugar, ol. menth. pip., and ol. anethi, and a small quantity of laudanum.

Chlorodyne. Chloroform, ether, hydrocyanic acid, morphine, cannabis indica, capsicum, peppermint, treacle.

Clarke's Blood Mixture. The active constituent is potassium iodide (about six grains to the ounce).

Oxien. Powdered sugar and starch, and ol. gaultheriæ.

Ozerine. Potassium bromide and ammonium iodide with chloroform water.

Pink Pills. Iron sulphate, an alkaline carbonate, and liquorice, thickly coated with sugar and coloured with carmine.

Phospherine. Quinine, phosphates, and hypophosphites.

Seigel's Syrup. Aloes, capsicum, liquorice, and treacle.

Steedman's Teething Powders. Calomel and starch.

Warner's Safe Cure. Potassium nitrate (about ten grains to the ounce) and various diuretic herbs.

Woodward's Gripe Water. Liquor magnes. carb., ol. anethi, sugar, and a trace of alcohol.

The British Medical Association has issued a full list of Quack Remedies in book form, which should be consulted by those who have an interest in this subject.

A CHAPTER ON MEDICAL JURISPRUDENCE IN INDIA[1]

In this chapter attention is drawn to those aspects of medical jurisprudence which will more especially interest practitioners of medicine in India and other parts of the East. The facts, figures, and experiences here noted do not come within the average experience of practitioners in temperate climates.

The medical witness in India is confronted with special difficulties in his attempt to get at the truth in criminal cases. These have been well summarised by Waddell.[2]

(1) Rapidity with which decomposition destroys dead bodies in the hot climate ;

(2) Facilities for concealing and destroying dead bodies, together with the general practice of rapid cremation or burial within a few hours after death ;

(3) Insufficient particulars of the crime in the police reports accompanying the alleged assaulted person, or a decomposed dead body ;

(4) Untrustworthiness of so much native evidence, owing to the wide prevalence of false swearing and fabricating false charges.

1. **Rapid decomposition** often renders the autopsy not only a difficult, but a very trying, operation for the medical officer. It is, nevertheless, his duty to perform it as thoroughly as possible. In some cases the result will be that no certain clue will be found ; in most, however, it is possible to determine at least the cause of death.

A dead body which has come into the hands of the police has to be carried in the hot weather, covered up with a cloth and wrapped in a piece of bamboo matting, for many miles—thirty, forty, or even sixty for a medico-legal examination. There may have been delay before the body was found, there almost certainly has been delay after the body was found, and it is easily understood how in the blazing sun or in the still Indian " hot weather " night, the process of decomposition may be well advanced before the body reaches the mortuary. It is comparatively rare, however, for a body to be too decomposed for the medical officer to be unable to find the cause of death. Even in the damp warm climate of Calcutta this plea is but seldom used. In a series of over thirteen hundred cases examined at the Alipore Morgue only thirteen were reported to be " too decomposed " for the purpose.

2. The second difficulty is **the rapid cremation or burial of bodies.** The bodies of Hindus who die are cremated, and the bodies of Mahometans are buried on the day of death. Even in cases of Europeans and in the cold weather it is seldom that a funeral takes place much later than twelve hours from the time of death. But besides these legitimate methods of disposal of bodies there are many others in India. Bodies are easily concealed in rivers, wells, swamps, pieces of jungle, or among the standing crops. In such places the body is soon found by jackals, crows, dogs, or in a river by crocodiles, fish, etc., and is soon mangled out of all recognition.

[1] This section was originally written by the late W. J. Buchanan, B.A., M.D., D.P.H., Major I.M.S.

[2] " Medical Jurisprudence for India," p. 24, ed. 1928.

3. Again, the police report which accompanies the body gives the medical officer a few particulars as to where the body was found, but it rarely gives him any clue to the cause of the injuries, and the vague statements of "believed to have been beaten," or such-like, are often contradictory and not improbably false, or, to put it mildly, vague and misleading.

4. The fourth difficulty is **the falseness of much of the evidence** given by natives of India. It is a disputable point how much of this is due to "inherent Oriental deceit," as one author puts it, or to fear, stupidity, apathy, or malice, or to the fact that the witnesses have been "tutored" by the police. Such false charges are generally supported by a wonderfully minute direct or circumstantial evidence; in fact, it is often owing to the evidence being too good that it is suspected and found to be false.

It would be a great mistake to imagine that the Indian criminal is a monster of iniquity; the vast majority of them are mild and somewhat simple men. In the Punjab and on the frontier the violent criminal is to be found, but the "habitual" criminal of an Indian prison is usually merely a habitual thief. Murderers are often mild and inoffensive-looking men, and indeed they are very frequently what the school of Lombroso calls "criminals by passion"; that is, they have attacked or assaulted their victim not as the result of long premeditation, but in a moment of passion, roused by some outrage done by the victim to some member of the murderer's family. Any large prison in India will be able to produce a dozen or more such cases, and they are generally well-behaved and quiet in prison. There are, however, other cases where the moral insensibility displayed is so inhuman and unnatural that in the European mind the utmost indignation is aroused.

THE ONSET OF CADAVERIC CHANGES IN INDIA

The classical experiments on this point were undertaken in 1883 in Calcutta by Dr. Coull Mackenzie, for many years police surgeon in Calcutta.

It is obvious that it may often be a matter of the greatest importance to ascertain the hour of death. Hence a study of the time of onset of cadaveric change becomes a necessity for the medico-legal expert.

Mackenzie's observations were made in a large hospital in Calcutta in the months of July, August, and September (average aerial temperature 85·5° F.), and in October (average temperature 81° F.).[1] The bodies were those of patients who had died of chronic disease in the hospital. The following is a summary of the observations made by Mackenzie[2]:—

Changes, onset of	Averages		Earliest		Latest	
	hrs.	mins.	hrs.	mins.	hrs.	mins.
Muscular irritability lasts from death	1	51	0	31	4	30
Rigor mortis begins	1	56	0	40	7	0
Ditto, duration of	19	12	3	0	40	0
Green discoloration appears	26	4	7	10	41	0
Ova of flies appear	25	57	3	20	41	30
Moving maggots appear	39	43	24	18	76	0
Vesications appear	49	34	35	0	72	0
Evolution of gases	18	17	5	50	34	30

[1] *Ind. Med. Gaz.*, 1889, p. 167, etc.
[2] Waddell's "Medical Jurisprudence," 1928, p. 92.

These observations may be compared with those of Assistant Surgeon Purna Chander Singh, of the Patna Medical School, which are reported in detail in the *Indian Medical Gazette* for June, 1902, p. 234. These observations were made in the very hot months of May, June, and July at Patna, a very hot place :—

Case 1. Female, aged forty-two. Body examined twenty and a half hours after death. Body swollen ; vesicles on arms, neck, and chest ; brain soft and pulpy ; vesicles on surface of lungs, liver, kidneys, and uterus decomposed. Date 9th May, 1901. Maximum temperature, 105° F. ; minimum, 79° F.

Case 2. Hindu male, aged thirty, died of assault. Body examined fifteen hours after death. Abdomen distended ; brain soft ; other organs slightly decomposed. Maximum temperature, 85° F. ; minimum, 71·5° F. Date 21st October, 1900.

Case 3. Hindu male, aged forty, beaten to death. Body examined twenty-six hours after death. Abdomen distended ; vesication on leg and thigh ; brain soft ; lungs, heart, etc., discoloured and softened. Date 30th May. Maximum temperature, 86° F. ; minimum, 76° F.

Case 4. Hindu male, aged forty, died of fracture of skull. Body examined forty hours after death. Cuticle detached ; bullæ on different parts ; eyes swollen ; tongue protruded ; fæces had escaped from anus ; hair of head easily detached ; brain reddish, soft or pulpy ; lungs decomposed ; blebs on their surface ; liver, spleen, etc., soft and decomposed. Date 2nd July. Maximum temperature, 92° F. ; minimum, 77° F.

Case 5. Hindu male, aged forty-five. Body examined forty-one hours after death. Body swollen ; blebs ; cuticle detached ; fæces escaped ; brain very soft, and could not be removed entire ; all organs decomposed. Date 1st September. Maximum temperature, 88·3° F.

Case 6. Hindu male, aged thirty-five, died of peritonitis after an assault. Body examined thirty-two hours after death. Body swollen ; blebs on abdomen ; face swollen ; tongue protruding ; fæces escaped ; all organs discoloured and softened. Date 15th March. Maximum temperature 85° F. ; minimum, 57° F. ; mean, 69° F.

The writer adds :

" The soft pulpy condition of the brain, on account of which the organ could not be removed entire from the skull, occurred in Patna within twenty and a half hours of death in May, within thirty-one hours in June, thirty-seven hours in July, and forty-one hours in September. It may be noted that May is the hottest month in the year at Patna, as from June to October the rains sensibly modify the fierceness of the heat."

These observations therefore show that decomposition sets in early in the hot weather in India, and advances with a rapidity quite out of the experience of European medical jurists.[1]

Under certain conditions putrefaction and decomposition may not take place ; but the body may undergo the *post-mortem* change known as saponification.

SAPONIFICATION, WITH FORMATION OF ADIPOCERE

This has already been fully discussed.[2] The following cases, however, should remain for reference.

Some years ago Mackenzie, then police surgeon of Calcutta, published eight cases, and Buchanan collected three more bearing on this question.

The e even cases are, briefly, as follows :—

1. A body was found in an advanced state of saponification on removal from a tank where it had laid for " several days."

[1] *Vide* Vol. I., pp. 228 *et seq.*
[2] *Vide* Vol. I., p. 253.

2. The body of a groom, exhumed from a damp Mahometan burial-ground four days and four hours after interment, was found to be in an advanced state of saponification.

3. A Chinawoman disinterred seventy-six hours after burial was also found in an advanced state of saponification.

4. A Bengali was drowned in the river Hoogli, the body was recovered after three days, and the internal organs were found saponified.

5. The body of a European, two days in the water, was examined, and all the external portions of the body were found to be saponified.

6. The body of a European sailor was recovered from the river eight days and ten hours after drowning. The external parts, the heart, liver and spleen, were found saponified.

7. The body of a sailor recovered from the river on the fifteenth day was found to be in an advanced state of saponification.

8. The body of a European youth was recovered after having been in the river seven days ; it was in an advanced state of saponification.

9. D. M. Moir's case. A body was exhumed after having lain in a damp grave, at the depth of three feet, on the side of a lake. The body was so much saponified that Moir was able to confirm completely the previous *post-mortem* examination. The soil in which the grave had been dug was damp, being saturated with the rain of the previous three months' monsoon.

10. The tenth case is recorded by Dr. R. S. Ashe. The body was that of a boy, aged nine, exhumed four days after burial. The skin of the abdomen, chest, and extremities was found to be mottled and waxy looking, and free from offensive odour. Portions were sent to the chemical examiner, Calcutta, who reported that "partial saponification had taken place in the tissues." This opinion was also confirmed by the Professor of Pathology at the Medical College, to whom the specimens were also submitted for opinion.

11. The assistant surgeon s.s. *Mitra* informed Gibbons that in December, 1897, he had examined a case of saponification of the entire body, which from the history must have been buried within three months.

MUMMIFICATION,

or the desiccation of the body, may also occur in India, not in the damp heat of Lower Bengal, but in the dry sandy deserts of Upper India.

AGE

The only published records bearing on this point which are specially derived from observations on natives of India are those by Powell published in the June issue of the *Indian Medical Gazette*, 1902.[3]

Powell also remarks : "The first permanent molars appear with great regularity in the sixth or seventh year. Of forty-one children aged seven, all had their first permanent molars. . . . All nine-year-old children, natives, Jews, and Parsees, had had all their permanent incisors." The canines show greater variation. They usually appear in the eleventh to twelfth year, but Powell has seen permanent canines in a child of nine. The second molars come with great regularity in the eleventh or twelfth year. He has "never seen a Hindu or Mussulman child of twelve without the second molars." He has also seen wisdom teeth as early as eighteen years and two months.

RELATION OF HEIGHT AND WEIGHT

There are no observations published on the relations of height and weight according to age for the peoples of India. The following table, compiled by Buchanan from weights and measurements of 28,000 male adult prisoners admitted into Bhagulpore Central Gaol, may be given

[1] Powell's table of dentition closely approximates to that given in Vol. I.

It is closely allied to a similar table which was compiled on the same lines at Nagpur, in the Central Provinces, and may be taken to represent the average height for weight of the peasant population of the part of Bengal known as Bihar :—

Height	Weight	Number of Observations
5 ft. 0 in.	100 lbs.	1,863
5 „ 1 „	102 „	2,059
5 „ 2 „	106 „	5,226
5 „ 3 „	109 „	5,787
5 „ 4 „	112 „	6,107
5 „ 5 „	115 „	3,040
5 „ 6 „	118 „	2,498
5 „ 7 „	121 „	1,389
5 „ 8 „	125 „	623
5 „ 9 „	129 „	220

The average weight of Indian children at birth has been estimated as 5½ lbs.

The average weight of a Bihar peasant may be taken to be 110 lbs., and his average height 5 ft. 3 in.

What has been called in India " Buchanan's formula " for calculating weight for height is as follows :—

" Taking 5 ft. as equal to 100 lbs., add 3 lbs. in weight for every inch above 5 ft., *e.g.*, 5 ft. 5 in. $= 100 + (3 \times 5) = 115$ lbs."

In the case of men over 5 ft. 8 in. in height it was found that the addition of 4 lbs. for each inch gave a weight which was not far from correct.

RACIAL DIFFERENCES IN THE SKELETON

This has been worked out in detail by Dr. Havelock Charles, of the I.M.S., Professor of Anatomy in the Calcutta Medical College. His observations [1] were, it is understood, made in the dissecting room of the Medical School at Lahore, in the Punjab. These might well be of importance in the case of the disappearance of a European in India, and the production of bones by the police :—

Spinal Column. " As a rule the body of a Punjabi's lumbar vertebra is thicker behind than in front. As the bone matures with age the excess of the posterior over the anterior depth becomes more pronounced.

" In the female the anterior measurement is greater than the posterior. . . .

" The fifth lumbar vertebra is only exceptionally wedge-shaped, as in the European.

" Up to the age of twelve none of the typical changes have taken place, but the deepest part of the body of a lumbar vertebra is behind and not, as in the European, in front."

The lumbar curve is straight or very slightly convex. Ninety-six is the index for the European lumbar curve (Turner), 106·8 for the native of the Punjab.

[1] Published in " Transactions of Indian Medical Congress," 1884.

The accessory processes of the fifth lumbar vertebra are frequently largely developed in the native, and often articulate with the alæ of the sacrum.

The articular surfaces of the sacrum in 78·7 per cent. of cases is formed of only two vertebra. In European sacra this is formed of three vertebræ (Macalister).

Acetabulum. The differences are :—

1. In natives of India the ischial portion of the facies lunata is very large. The rim of the acetabulum here is very prominent, and the groove for the obturator external muscle is consequently deep.

2. The extension forwards and the widening out of the lower horn of the facies lunata, whereby the cotyloid notch is, as it were, partly bridged over instead of being an irregular open space. It looks as if the transverse ligament were ossified on its ischial side.

3. The cotyloid notch, which in the European bone is, as a rule, open, presents in well-marked Indian bones the characteristic of being arched over by the forward and upward prolongation of the inferior cornu of the facies lunata. The superficial boundary of the cotyloid notch in Europeans consists of the transverse ligament alone ; the same boundary in the Indian consists of bone (part of the ischium plus the transverse ligament).

The Head of the Femur. The articular area is of greater extent relatively and absolutely than that of a European bone. The surface is specially prolonged to adapt itself to the modified facies lunata of the acetabulum during extreme flexion and partial abduction, and during semi-flexion and extreme abduction occurring at the hip-joint in the squatting and sartorial postures.

The Neck of the Femur is longer relatively than in the European.

The upper surface of the internal condyle of the femur is partly articular. This is not so in Europeans, where it is merely rough for the insertion of the gastrocnemius. It is due to the power of extreme flexion possessed by the Oriental knee joint.

The Head of the Tibia is set on the shaft very obliquely. An Oriental tibia can easily be held by the finger and thumb when the internal tuberosity is grasped behind by them.

The upper surface of the internal tuberosity slopes considerably downwards and inwards ; it is never flat, as in European bones.

The external tuberosity of the tibia has its condylar surface convex from before backwards, and the articular surface is well prolonged downwards posteriorly.

The upper part of the tibial diaphysis is commonly directed obliquely backwards.

On the anterior margin of the **lower extremity of the tibia** a facet will be found in most cases on what is the ligamentous area of the European bone.

In upwards of 17 per cent. of tibiæ a second facet on the same border, but occupying a more internal position, will be seen. Both these articulate with corresponding articular areas on the upper surface of the neck of the astragalus.

The Astragalus differs considerably from the European. There is a facet on the upper surface of the neck to the outer side ; there is a facet on the same surface more internally, which is continuous posteriorly with the trochlea, and internally with the pyriform malleolar surface.

The outer margin of the neck of the astragalus is markedly thinner than in the European bone. *The Under-surface.* In the European bone the deep concavity, or articulation with the large convex facet on the upper surface of the os calcis, is generally bounded by two sharp non-articular margins. In Oriental bones the outer margin is frequently articular on its inferior aspect, as this part, when the facet exists, articulates with the upper surface of the greater process of the os calcis.

The Os Calcis. On the upper surface of the great process there is often amongst Orientals a facet continuous with the larger posterior articular surface of the body of the bone, whereas this surface is rough in European bones.

The Skull. For practical purposes it may be assumed that most male Indian skulls have a cubic capacity of 1,360 cc. or under, whereas the European male skull runs to 1,500 cc. and upwards. The measurement of the cranial capacity can be made by mustard seed, easily procurable, when small shot is not available. Dr. Charles also points out that it is rash to assume that the bones of an Oriental are necessarily smaller than those of a European. Many large, tall "up countrymen" have large bones.

WEIGHT OF VISCERA

The following table was compiled by Buchanan from his own observations, and others made by Dr. R. Maddox, I.M.S., upon adult male prisoners in Bengal, who had died of disease in prison :—

NATIVE MALES

	Number of Cases	Average Weight	Highest	Lowest	Causes of Death
		Oz.	Oz.	Oz.	
Liver	333	44	108	13	—
Spleen	314	10½	64*	1	*Malaria
Lung, right	224	16	52†	5	†Pneumonia
,, left	224	14½	43	5	—
Heart	238	7½	20‡	4	‡Apoplexy
Kidney, right	246	3¾	8	2	—
,, left	246	3¾	8	2	—
Brain	143	44	56	33	—

NATIVE FEMALES

	Number of Cases	Average Weight	Highest	Lowest
		Oz.	Oz.	Oz.
Liver	88	37½	62	16
Spleen	91	6½	48	1
Lung, right	49	9½	20	6
,, left	49	9½	17	4
Heart	46	6	9	4
Kidney, right	68	3½	6	1
,, left	68	3½	6	1
Brain	7	37	42	26

VIOLENT DEATHS

The largest number are due to accident ; then comes suicide, and lastly homicide.

Suicide

Dr. Kenneth McLeod published a valuable monograph on suicide in India. From this it appears that in Bengal and the Punjab the reported average annual suicide rate for the five years which ended 1876 was in Bengal 31·3 and Punjab 12·7 per million of the population ; in Madras it was 62 for males and 68 for females ; in the United Provinces of Agra and Oudh 27·8 for males and 71 for females ; in Calcutta it was, males 64, and females 104 per million. In India the female suicide rate always exceeds that of males, the reverse being the case in England.

Kenneth McLeod also gave the following table on the various methods of suicides per thousand of the two sexes *in Calcutta* :—

Method	Males per thousand	Females per thousand
Hanging	179	346
Drowning	127	54
Poison	547	562
Cuts and Stabs	59	16
Gunshot	37	—
Otherwise	51	22

From this it is seen that in that city the favourite means of suicide was poison, but this does not apply to other parts of the country.

The following table is compiled from four provinces in India :—

Methods of Suicide in India per Thousand Suicides in each Sex.

Method	Males	Females
Hanging	368	278
Drowning	354	576
Poison	168	119
Cuts and Stabs	65	11
Gunshot	25	—
Otherwise	20	16

From this it will be seen that male would-be suicides prefer first hanging, then drowning, and then poison, while females far and away prefer drowning, then hanging, and next poison.

The chief causes of suicide are shame, grief, physical suffering, revenge (Chevers), and from religious motives, as in the now abolished [1] custom of *sati*, where the widow threw herself on the funeral pyre of her deceased husband.

Homicide

The causes which lead a man to murder another are often in India trivial in themselves : quarrels about land, murder for the sake of robbery, such as the ancient *thuggi* and the more modern form of *datura* poisoning, and murder arising from outrages done to husband or to wife.

[1] Illegal—a trial arose on a case in 1905 (*vide Lancet*, February 18th, 1905).—Ed.

Infanticide

The special causes why infanticide is common in India are the restrictions placed upon the remarriage of Hindu widows. Such women are deprived of their husbands often at a very early age, and are exposed to temptations, without any choice of remarriage. This leads to the destruction of the offspring of illicit intercourse. Among certain classes in India it is considered necessary to have all the daughters of a family married. This is not always possible, and is always expensive ; hence a certain amount of female infanticide is practised.

Chevers has recorded a case where, because three successive female children were born, the father exposed the last born child in the jungle. Chevers also records that in a special inquiry ordered by Government into the practice it was found that in twenty-six villages out of 308 no female child under the age of six existed. In another Rajput village 104 boys and only one girl were found, in another 284 boys but only twenty-three girls.

CRIMINAL ABORTION

Criminal abortion is resorted to in most countries, but is said to be very common in India and is largely due to the non-remarriage of Hindu widows.

The methods of producing abortion in India are usually crude, and frequently lead to loss of life. It is impossible to obtain statistics of the degree of prevalence of this offence, as it is only the fatal cases that come to the notice of the police. If the case recovers, the pyrexia is attributed to malaria, and if the woman dies, and a complaint is not immediately lodged, the body is burned or thrown into the river.

The methods are either mechanical or medicinal.

Mechanical. A very common method is by the use of "abortion sticks." Those commonly used are twigs of *Plumbago Rosea*, *lal chitra* in the vernacular, or of the oleander, *Nerium Odorum*. These sticks are five or six inches long, and are passed into the uterus either through the membranes, or between them and the wall of the uterus. It not infrequently happens that the wall of the uterus is thereby perforated, with the result that peritonitis supervenes and death follows.

In other cases the "stick" used is not in itself an irritant, but it is smeared with some irritant substance, as the juice of marking nuts (vernacular *bhela*), *Semecarpus Anacardium ;* the juice of jequirity (vernacular *rati*), *Abrus Precatorius*, or *madar*, the milky juice of *Calotropis Gigantea*, a common wild plant in India.

Medicinal. By the use of many indigenous drugs, which are credited with ecbolic or emmenagogue properties, as papaya seeds (*Carica Papaya*), carrot seeds, aloes, croton oil, mercury, quicklime, copper, or even the juice of bamboo leaves. Not infrequently a combination of more than one method is used, as indeed is the custom in countries other than India.

INJURIES IN GENERAL

One of the most common weapons in use in India is the *lathi*, or bamboo staff, frequently carried by all natives of India. It is often bound with iron or brass, and in the hands of a feeble man it is and should be regarded as a "*deadly weapon*." In 1872, Harvey stated that 32 per cent. of all medico-legal cases in Bengal were due to injuries

by blows of a *lathi*. In 1901, in the Arrah district alone, Stevens has recorded that, out of 223 police cases, in 204 the injuries were caused by *lathi* blows, including twenty-four fractures of bones.

There is one form of severe contusion of the chest which is worth mentioning, as it is very likely to mislead the young medical officer. It is described by Chevers, and is known in the vernacular as *bansdola*, or bamboo crushing. A bamboo is placed across the victim's chest. Men sit on the ends of it, and roll it backwards and forwards over the chest. The torture is horrible, the muscles are bruised, but in practised hands *no external mark is caused*, though on *post-mortem* examination the ribs may be broken and the lungs lacerated. This torture may also be applied to the limbs, or the torturer may kneel with his two knees in the two "Scarpa's triangles" of the victim. Here also considerable local damage may be done to the vessels and muscles without any visible external marks.

INJURY TO ORGANS OF THE BODY

The Spleen

There is no special Indian experience on injury to, or rupture of, any organ of the body except the spleen.

During the past years a considerable amount of evidence has been forthcoming which shows that the enlargement of the spleen, which is so common in India, is the result of infection by what is known as "the Leishman-Donovan bodies," and as these have been shown by L. Rogers to be stages in the development of a trypanosome, it is clear that what has been called malarial enlargement of the spleen, or tropical splenomegaly, should be considered as a result of trypanosomiasis. It is probable, however, that repeated attacks of any malarial fever can *also* produce enlargement of the spleen, for it is certainly a very common sequel of such repeated attacks. From the medico-legal point of view the causation of the enlargement is, however, of small importance compared with its results, especially when they end in rupture.

Rupture of the Spleen is an injury which is rare in England, and, consequently, has received but scant attention from writers on medical jurisprudence in the British Isles. The reverse, however, is the case in India, where it is extremely common, and is constantly appearing in the law courts as the cause of death. In fact, so common is it, that in the case of the sudden death of a native it might often be safely presumed that this was the cause. In the majority of those unfortunate cases in which a European is charged with having caused the death of a native by a blow or a kick, it is almost invariably found that the spleen was ruptured from a degree of violence which would have had no effect on a healthy person. A hostile newspaper went so far as to state that it did not believe in the existence of such an injury as rupture of the spleen—a statement based, I need hardly say, on the most absolute ignorance of the whole subject.

Rupture of the spleen, therefore, is a matter of the very greatest importance to the medical man practising in India, or in any other malarious country.

We have no statistics on a large scale as to the exact degree of the prevalence of enlargement of the spleen among the people of India, but such as have been compiled go to show that it is very considerable. Indeed, till recently an enumeration of the proportion of enlarged spleens in any community was used as a test of the malarial character of any locality.

In the European, the books on anatomy tell us, the spleen weighs from five to seven ounces. This is for Europeans whose average weight is usually taken at 150 pounds. The average weight of the native Bengalis, as the result of some 28,000 observations, is about 110 pounds only; but in them the spleen was found to weigh on the average ten ounces (average of 314 careful records). The largest weighed was sixty-four ounces, that is heavier than an ordinary liver, but many larger than this have been found.

It is obvious that a large spongy organ, taking up much more than its proper space in the abdomen, is peculiarly liable to external injury and that, owing to the friability of the organ and the large quantity of blood it contains, such injury must almost always be fatal.

In an admirable article,[1] Dr. D. G. Crawford has analysed very thoroughly a series of 304 cases of ruptured spleen. These 304 cases were all taken from records of *post-mortem* examinations made for medico-legal purposes, and out of over 9,000 such records examined, the number of cases of ruptured spleen amounted to not less than 3 per cent. of the whole. Moreover, out of these 9,000 cases the spleen is noted as enlarged in no less than 37 per cent., and in some districts well over 50 per cent. of the spleens are recorded as enlarged. This means that more than one-half of the persons whose bodies come to be examined for the purposes of justice have enlarged spleens.

Following Crawford, we may discuss this question under several headings :—

Age and Sex. Examination of statistics shows that there is but little difference between men and women in their liability to this injury : as regards age, nearly two-thirds were adults, that is, persons of an age most likely to be engaged in fights and quarrels.

The Cause of the Rupture. Of the 304 cases, omitting 57 in which the cause is recorded as unknown and a few from miscellaneous causes to be mentioned below, we find 102 due to blows from sticks, 62 to blows of the fists or kicks, 22 to falls, chiefly out of trees, 2 to pressure on the body (a familiar form of torture), 23 as part of a murderous assault, and 20 to being run over or to a heavy weight falling on the body.

The miscellaneous causes recorded are of importance, as showing what a slight degree of injury may lead to a fatal result. Among them were a clod of earth thrown and striking the left side, the blow of a shoe, or a wooden stool, or the prod of a cow's horn. Others were, being knocked down (not run over) by a horse, the blow of a fall to the ground, or the kick of a horse.

The following cases may also be quoted :—

A European gentleman slipped on the floor of his bathroom, and died within a few minutes. At the autopsy the spleen was found to be ruptured and to weigh nineteen ounces. Even this simple fall caused no less than four lines of rupture.

[1] *Indian Med. Gaz.*, June, 1902.

James has recorded the case of a shepherd boy who, while indulging in horse-play with another youth, fell and died within three hours. The spleen was found to weigh twenty-two ounces and to be ruptured in two places on the lower surface. In another case a Punjabi boy died after a blow which was ascertained to have been on the right side. The spleen was found enlarged, with a rupture one and a half inches long on the inner surface. James has also recorded another remarkable case of what he calls " spontaneous rupture " of this organ in the person of a Punjabi who, while conducting his own case in a law court, fell down suddenly. Not the slightest evidence could be obtained that he struck anything as he fell, but at the autopsy it was found that the spleen was much enlarged (weighing no less than three pounds thirteen ounces), and ruptured for six inches along its inner surface.

Two other cases of " spontaneous rupture " of the spleen have been published.[1] These cases show that when the organ is diseased, the slightest blow is sufficient to cause its fatal rupture, if indeed muscular action may not be by itself sufficient.

The Site of Rupture. Of 262 cases where the site of the rupture was noted, 133 were on the inner surface, 55 on the outer surface, and 116 either on two surfaces or irregularly distributed. Of 304 cases 225 were single ruptures, and 79 were multiple. It appears therefore that the inner surface is by far the most commonly ruptured, and it is said that on this aspect the spleen capsule is thinnest.

In all the above cases the spleen was recorded as more or less enlarged ; but there were eight cases in which at the autopsy it was recorded as not enlarged. In these eight cases the injury was either severe or multiple (murderous assault or " run over ").

A prisoner in a gaol either fell on some bricks or was roughly handled by another prisoner (the evidence was not decisive). At any rate the spleen was found ruptured, and it was described by the reporter as " healthy " ; it was neither markedly diseased nor enlarged.

It is, perhaps, worth noting that in five of these eight cases the stomach is stated to have been found full, and in only one case it is said that the stomach was empty. Crawford, however, who has investigated this point, is of opinion that there can be found no very definite connection between rupture of the spleen and a state of fulness of the stomach.

Complications. Of course, in cases of great violence it is natural to expect damage to other organs, but an analysis of Crawford's figures shows that in only thirty-two cases (10 per cent.) was any other organ than the spleen ruptured. In nineteen of these thirty-two the liver was also ruptured.

The Period of Survival after Rupture. This is often a most important legal question. We may quote a few cases bearing on this point. In Russell's " Malaria : its Causes and Effects," a case is related in which a man received a severe injury to the spleen and recovered ; but the injury to the spleen was confirmed some years after, when a *post mortem* examination took place on his body. Buchanan was able to collect seven cases of survival for considerable periods after undoubted rupture of the spleen.

[1] *Indian Med. Gaz.*, 1904.

In four cases the victims survived over twenty-four hours, in one case for five days, in two cases for four days, in another case for two and a half days, in another a "few days," in another for three days. The longest period of survival in this series is that of a man admitted to a Calcutta hospital with a rupture of the spleen: he remained there for seventeen days, and the injury was confirmed *post-mortem*.

In some cases the period of survival is passed in unconsciousness, but in others there can be no doubt that the patient may be able to speak or make a dying declaration, etc., points often of the greatest legal importance.

A question may arise as to the possibility of a man with ruptured spleen being able to get up and walk a certain distance. This point is not often noted in recorded cases, but in reading them one frequently finds nothing that makes such impossible. Doubt is set at rest by the following case, published in 1867, by Dr. Hutchinson:—

> An old man, after having been severely beaten by a bamboo, walked to his home, a distance of about half a mile, and there died almost immediately. The *post-mortem* examination showed that the seventh and eighth ribs on each side had been fractured. The spleen was ruptured, and also the liver.

It is worth adding that a case has been published,[1] in which at an autopsy on a Sepoy, aged twenty-four, there was found a total (congenital) absence of the spleen, along with a transposition of all the abdominal and thoracic viscera.

Chevers has pointed out that rupture may occur in (1) simple engorged spleen, (2) enlarged engorged spleen, (3) the small hard fibrous spleen, and (4) the large hard fibrous spleen.

The so-called "ague cake" spleen is enlarged and often so fibrous and hard that it seems at first sight little liable to rupture, but in such cases a recent attack of fever may have produced engorgement, which would have the effect of making it softer and more friable.

THE DURATION OF DIGESTION OF INDIAN FOODS

Experiments to show the periods required for the digestion of foods have usually dealt with European foods, *i.e.*, meat, bread, and vegetables, and Dr. Beaumont's classical experiments still remain unchallenged, but it is to be noted that the periods given do not refer to the digestion of Indian foods in Indian stomachs, on which but few observations or experiments have been recorded, hence the value of the observations below.

The natives of India use either rice, wheat, or other grains as their staple food. In addition to this many use some of the pulses, and comparatively few eat meat, fowls, or fish. The people of Eastern Bengal, however, take a small quantity of fish, if they can procure it, at both of the chief meals of the day. Rice is largely consumed by the inhabitants of Bengal, Madras, and Burma, along with vegetables, fish, and pulses. In the United Provinces, and in the Punjab, rice is but little used. The people there eat chiefly wheat, maize, millet, and such cereals, cooked into unleavened cakes, or *chapattics*. The rice-eaters take a large quantity of this grain. Cooked rice weighs almost three times as much as dry rice, and an ordinary meal of a fairly well-to-do Bengal native consists of about twelve ounces of dry (about thirty-six ounces cooked) rice, a

[1] *Indian Med. Gaz.*, November, 1903, p. 417.

couple of ounces of dry pulse or fish, a few ounces of watery vegetables, and not less than a pint of water. The bulk of this is very great, and it is probable that the routine use of pan (betel) and other digestives is due to their power of aiding the digestion of such a bulk. It is obvious that it may be of the greatest medico-legal importance to know the exact time at which a deceased person died. One method of estimating this is by the state of digestion of the food in the stomach, presuming that the hour at which deceased took his last meal is known.

The following observations of Dr. P. C. Singh, of Patna, have been published. They were made on bodies sent in for medico-legal examination :—

1. A Hindu, aged thirty-five, took food at 8 a.m. He was severely assaulted at ten o'clock (two hours later), and died at 2 p.m. from the effects of ruptured spleen. At the autopsy a large mass of undigested rice and pulse was found in the stomach. Death had taken place six hours after the last meal ; but it is possible that the process of digestion may have been interrupted by the shock and hæmorrhage at ten o'clock.

2. A young man took food at 11 p.m. ; he had an epileptic fit at 2 a.m., and died at 5 p.m. the next day. The stomach was found half full of undigested rice.

3. A man took his evening meal at 10 p.m., went to sleep soon after, was murdered in his bed at 5 a.m. (*i.e.*, seven hours after taking food). A small mass of undigested rice and potato was found, so that stomach digestion was not completed even in seven hours.

The following experimental observations were made by washing out the stomachs of healthy persons at fixed periods after taking food :—

1. Large meal of pulses, rice, and vegetables at 12 noon, stomach washed out after three hours. Some undigested rice remained.

2. Same person another day, stomach washed out after four hours. One ounce of undigested rice was recovered.

3. Same person another day. After five hours some undigested rice flowed out from tube (250 grains counted), and so up to seven hours, when even then some undigested grains of rice remained in the stomach.

4. A similar experiment on another man. Some 200 grains of rice, undigested, were found on washing out after five hours.

5. Similar experiment. Two drachms of undigested rice were found after six hours.

6. Same person fed on rice and chapatti (a sort of unleavened bread in the form universally used by up-country natives). After six hours some rice and a piece of the chapatti were found undigested.

In two similar experiments pieces of the chapatti, undigested, were found in the stomach after so long intervals as six hours and thirty minutes, and after six hours and forty minutes.

These experiments and observations seem to show that some portion of a meal of rice, pulses, etc., may be found undigested even six or seven hours after the taking of food.

INDIAN HEMP

In the year 1893, the Government of India appointed a commission to inquire into the cultivation of the hemp plant, the preparation of drugs from it, and the effect of their consumption on the social and moral condition of the people. This commission visited all parts of India, made elaborate and thorough inquiries, and published the results of its investigations in a report which, with the records of evidence, etc., consists of seven large volumes. In this report is contained all that is known about the hemp drugs, their use and abuse, in India.

The two plants *Cannabis Satira* and *Cannabis Indica* are apparently identical, "the Indian plant being viewed as an Asiatic condition of the species." There are no botanical characters to separate them. Though

the hemp plant is believed by the most competent Indian botanists to have been introduced from Central Asia, and not to be an indigenous plant, yet it now grows wild over an immense area throughout the Himalayas from Kashmir to the extreme east of Assam.

The products of the hemp plant which concern us from a medico-legal point of view are **ganja, charas** and **bhang.** Though these names are in some places confused or mixed, yet, generally speaking, the following definitions given by Prain, of the Calcutta Botanic Gardens, hold good :—

" *Ganga* consists of the dried flowering tops of cultivated female hemp plants, which have become coated with resin, in consequence of having been unable to set seeds freely.

" *Charas* is the name applied to the resinous matter which forms the active principle when collected separately.

" *Bhang, siddhi, sabsi, patti*, are different names applied to the dry leaves of the hemp plant, whether male or female, cultivated or uncultivated."

It must, however, be noted that the word *bhang* is commonly used for the liquid form in which the hemp drugs are consumed ; *i.e.*, *ganja* pounded up and made into drink becomes the " bhang " of ordinary parlance.

The hemp drugs may be smoked, drunk, or eaten.

Smoking. Ganja is chiefly used in Bengal. The ordinary method in Bengal is as follows : A small quantity of ganja is taken, one sixteenth of a lota, and is kneaded in the palm of the left hand with the thumb of the right, a few drops of water being added if necessary. When it ceases to give any colour to the water it is ready to be smoked. The pipe or *chillum* is a bowl with a short neck, the same as that commonly used by natives for smoking tobacco. First a small foundation of tobacco is put into the pipe. On this is placed the prepared *ganja*, which has been chopped up. Then comes another layer of tobacco, a bit of live coal is placed on the charged pipe, and a damp cloth is generally wrapped round the neck of it and folded into the palm of the left hand, while the pipe is grasped by the neck between the thumb and first finger. A few short breaths are blown and drawn to light the pipe, and when this is done one long deep draught is taken into the lungs. The pipe is then handed on to a companion and goes the round of the circle.

In the Punjab and United Provinces *charas* is chiefly smoked, but *charas* is only smoked in Bengal by well-to-do people, on account of its expense. In the provinces just mentioned the *charas* is mixed with tobacco in the form in which tobacco is generally used by natives in their *hukka*. The draught is always taken into the lungs.

Ganja may also be smoked in the hukka, and even cigarettes may be made of the leaves, but usually the leaves are used only by very poor people in this way.

Well-to-do people, however, are not content with the simple means of smoking, and they prefer to mix with the *ganja* or *charas* certain spices, such as musk, mace, saffron, cloves, cardamoms, or rose leaves, etc. Sometimes even more potent drugs are added, as opium and datura. The use of datura, opium, arsenic, or nux vomica is, however, confined to excessive consumers, to whom the simple drug has ceased to give the required degree of exhilaration or stupefaction.

Methods of **drinking** *Hemp Drugs.* As with smoking, so in the case of drinking, there is a simple or common form and other more elaborate compounds. The simple form is merely to pound the drug very fine with some black pepper, to add water (according to the desired strength of the drink) and filter the mixture through a cloth. For this purpose the leaves of the plant are generally used, or the leaves and the flower heads. This is the preparation commonly called in Bengal *siddhi* or *bhang*, and in other provinces usually *bhang*.

This drink may be fortified by the addition of harmless spices and perfumes, such as anise, fennel, coriander, dill or cardamoms, or to meet the craving of the excessive consumers, the drink may have datura seeds, opium, nux vomica, or arsenic added.

Eating. *Ganja* and *bhang* (*i.e.*, leaves) are also eaten mixed with any of the spices mentioned above, or with *gur* (crude molasses), and swallowed in the form of a bolus. The practice of thus eating the hemp drugs is, however, much less common than either smoking or drinking them.

Another preparation is in the form of sweetmeats, called *majun*. *Ganja* and *bhang* (leaves), and more rarely *charas*, are used, the basis of the sweetmeat being sugar, milk and spices.

Majun is largely used for certain Hindu feasts, and is evidently credited with aphrodisiac qualities.

The custom of offering an infusion of the leaves of the hemp plant to guests on religious festivals is almost universal in India. In fact, the use of *bhang* is intimately associated with many social and religious observances of the people, just as is the use of wine and spirits among European nations.

The above details have been given of the varieties and uses of these drugs, for considerable ignorance exists as to them, and it is not possible for a medical officer to make any inquiries as to their use in a case of insanity suspected to be due to this cause unless he has a clear notion of the customs of the people with regard to these drugs.

It may be remarked that there is a certain amount of popular prejudice against the smoking of these drugs. It is not quite respectable: at least respectable men do not smoke the drug in public, but usually in the privacy of their homes, and among friends and companions, and it is believed that on the whole smoking *ganja* or *charas* is more injurious than the drinking of *bhang*, but in considering the abuse of the drugs the question of adulterants, especially datura, must always be borne in mind.

In discussing the question, " Is the moderate use of drugs beneficial or at least harmless ? " we must clear our minds of prejudice. The fact is that the answer to such a question is the same as would be given by the average man to the same question about alcohol or tobacco. Neither alcohol, tobacco, nor hemp drugs are necessary. Moderation in their use does but little, excess certainly does much, harm.

It must also be remembered that hemp drugs are largely used for therapeutic purposes by Indian medical practitioners. They are often prescribed in bronchitis, asthma, and other respiratory complaints, and also as general and local anodynes. They possess a certain amount of

diuretic action, and though modern research does not favour the view that they are also aphrodisiacs, yet such is a popular belief in India. The probability is that, like alcohol, they give strength and free course to the predominant desires of the animal nature. On the whole the balance of evidence is that the *moderate* use of hemp drugs is not injurious.

As regards the formation of a habit, in this respect hemp drugs are like any other intoxicant, consumption tends to become habitual. The habit is strong, but the difficulty of breaking it off is not so great as in the case of alcohol or opium, but probably greater than that of the tobacco habit. The immoderate or excessive use of the drug is chiefly of importance in regard to its share in the causation of insanity.

The report of the Hemp Drugs Commission, from which we have quoted, has gone very thoroughly into the relation between hemp drugs and insanity. There is no doubt that in the records of Indian asylums there were, and still are, a great many cases which have been attributed to the excessive use of these drugs, but on examination of the records of cases in the asylums, the Commission proved clearly that, whatever may be the truth of the connection between hemp and insanity, the statistics of the asylums had no claim to be considered as trustworthy. It was proved in a large majority of cases that the entry of hemp drugs, under the heading of causation, in the asylum registers merely meant no more than that this "cause" was put down from the descriptive roll that accompanied the patient to the asylum, and that there was no better authority for the entry than that of the police subordinate who filled up the form. The Commission concluded that "excessive use indicates and intensifies mental instability, it tends to impair the mental faculties, and it may even lead to insanity." When taken in more than a moderate quantity, hemp drugs in any form produce a condition of intoxication, shown by laughing, singing, and other emotional actions. Hallucinations are produced, which are governed by the nature of the subject towards which the thoughts are most often directed. To the sensualist the effects are sensual, while the ascetic finds that his powers of contemplation are increased by a dose of hemp (Gibbons). In persons unaccustomed to its use the drug produces marked delirium with violence, or even mania. The stage of excitement lasts some hours; then the individual passes into a state of stupor (or, in rare cases, into fatal coma). After several hours recovery gradually takes place. The question then remains, are there good grounds for the popular belief (shared by many medical men) in a close connection between the abuse of hemp drugs and insanity? J. H. T. Walsh has ably discussed this in the *Journal of Medical Science*.[1]

His conclusions are as follows:—

"(1) That hemp drugs are very largely used in Bengal, smoked as *ganja* or *charas*, drunk as *bhang* or *siddhi*, or eaten as *majun*.

"(2) Among healthy persons *ganja*, smoked alone or with a very small addition of *datura* (two or three seeds), produces a condition varying from mild exhilaration to marked intoxication. The violent and intoxicating effects are less marked, or not seen at all, in persons having a regular and wholesome supply of food. Much the same may be said of *bhang*.

"(3) Among persons of weak mind, or with a marked neurotic tendency, even a moderate quantity or only a slight excess of hemp drugs may so increase the insanity, latent or evident, as to make such persons violent, morose, or melancholy.

[1] January, 1894.

according to the neuropathy with which we start. The presence of adulterations such as datura will increase these effects.

"(4) Abuse of hemp drugs, especially when adulterated with datura, will produce even in healthy persons a very violent intoxication simulating mania, or may lead to a morose melancholic condition, or to dementia. These conditions are generally of short duration, and the patient ultimately recovers. So common is absolute recovery that I think when a patient confined in an asylum for the treatment of insanity, said to be due to the abuse of hemp drugs, does not recover within ten months, these drugs were possibly only the exciting cause, and that we are dealing with an individual who was either insane previous to his use of intoxicating drugs, or with one in whom latent insanity has been roused into activity by the vitiating effects of excess of ganja, bhang, etc."

Running Amok. This is an expression applied to an impulsive act of reckless multiple homicide, without apparent or any real motive. The word *amok* is said to mean in the Malay language "frenzied." There is a popular impression that persons who "run amok" are in a condition of hemp intoxication. As shown above, no doubt abuse of hemp does produce a condition of violent mania-like intoxication, but there is little or nothing to show that the homicidal impulses of "amok" are in any way connected with the abuse of hemp drugs. No doubt murderers and thieves may often nerve themselves with hemp for the accomplishment of acts previously determined on, but this is only an Oriental form of "Dutch courage."

Chevers [1] quoted Dr. Oxley as dividing cases of amok into two classes: (1) cases where the motive is revenge for a real or supposed wrong, the assailant becoming perfectly reckless, and (2) the other form, very different and by no means so frequent. For instance, a man sitting quietly among his friends and relatives will without provocation suddenly start up weapon in hand and slay all within his reach. Dr. Oxley has known as many as eight killed and wounded by a very feeble individual in this manner. The next day when questioned why he did this he replied, "The devil entered into me; my eyes were darkened; I did not know what I was about." Dr. Oxley generally found these maniacs suffering from some gastric disease, these fearful ebullitions breaking out upon some exacerbation of the disorder. Dr. Oxley did not attribute this practice to the use of intoxicating drugs, and a more recent observer, Dr. J. D. Gimlette, agrees with this view. Gimlette considers "amok," as seen in Malays, to be pathological, and in some degree allied to the procursive form of epilepsy, in which the patient starts to run. There is always, he says (1) sudden paroxysmal homicide, generally in the male, with evident loss of self-control; (2) there is a preceding period of mental depression; (3) there is a fixed idea to persist in reckless killing, due to an irresistible impulse; and (4) there is a loss of memory of the whole attack.

POISONING IN INDIA

From times immemorial the use of poisons to remove objectionable persons has been very common in India. In a paper pleading for the restrictions of the free sale of poisons in India, Drs. Evans and C. L. Bose stated that "murder by poisoning is three times as prevalent, and suicide by poisoning many times more prevalent, in India than in England."

There is a great degree of sameness in the poisons used by the people of India for various crimes. For homicide, arsenic is by far the most commonly used, then come aconite and nux vomica, and (in cases of children) opium; for suicide, opium is used in a large majority of cases, then, much less frequently, arsenic; for stupefying previous to robbery, datura chiefly, and, more rarely, Indian hemp; for abortion, plumbago rosea and oleander.

[1] "Medical Jurisprudence," 3rd ed., Calcutta, p. 781.

In the present chapter only the most important poisons used in India are dealt with.

Arsenic

There are many reasons why arsenic should be the chief poison used for homicide. In the first place, it is easy to obtain, for white arsenic may be purchased in every bazaar, as it has its legitimate uses—*e.g.*, as a preservative of wooden posts againsts the attacks of white ants, in the making of leather, and in curing hides and skins. It is also largely used for destroying vermin, and as a medicine in the treatment of syphilis and the more chronic forms of the malarial fevers. Secondly, the acute effects of the poison so much resemble an attack of Asiatic cholera that suspicion is unlikely to arise, especially if it happens that cholera is at all prevalent in the neighbourhood at the time.

All forms of arsenic may be used for criminal purposes, but white arsenic, arsenious oxide (As_2O_3), is the form most commonly employed. Its colour is white : it has but little taste when mixed with sugar, sweetmeats, bread, or rice, in which vehicles it is usually administered. White arsenic is called in the vernacular languages *somul* or *sumbhul*.

The sulphides of arsenic are less commonly used for the purposes of crime in India. It is curious, too, that a very large proportion of the cases in which the sulphides have been used have been suicidal. In other instances arsenious oxide has been found mixed with the sulphates of iron and copper, and with the sulphide of mercury.

An important point in the criminal use of this drug, and one which often leads to its detection, is the enormous dose usually administered by the criminal to the victim. There are many cases in which the quantity is so great that it may be scraped off the walls of the stomach with a knife.

The motives which lead to the use of arsenic for homicidal purposes are chiefly revenge and sexual passion. Husband poisoning is commonly effected by the use of arsenic, and in some cases it is certain that the powerful drug was only used as a "love philtre," or as an aphrodisiac, and with no criminal intent. It is also an undoubted fact that in times of cholera prevalence arsenic is used as a means of getting rid of an enemy, or a rival in many disputes into which the land hunger of the Bengal peasant leads him.

Arsenic is less commonly used as an abortifacient, and usually with disastrous results. In such cases it is commonly applied as a mass of paste to the os uteri. It is seldom used for suicidal purposes, but when so used it is in very large doses—as much as 300 grains have been recovered in such a case, though, as said above, the mere fact of such enormous doses by no means negatives a homicidal view of the case.

Cases of accidental poisoning are not infrequent, owing to the common use of arsenic in the arts and as a medicine. Buchanan saw a case in which a native gentleman, who suffered much from fever, and could, or would, not take quinine, accidentally poisoned himself by the continued use of Fowler's solution in very large doses (40 to 80 minims).

Arsenic is very largely used as a cattle poison, though in the United Provinces its place is taken by the use of dried snake poison inserted under the skin on a piece of sharpened iron or wood.

In a few rare cases in which death from shock has resulted the stomach has shown no signs of congestion, and has even contained a large quantity of solid and liquid food, vomiting not having occurred.

In a series of 191 cases of arsenic poisoning four were recorded in which death took place within two hours, and in none of these was any congestion found. It would seem as if more than two hours' contact were required to produce the appearances of congestion.

As the decomposition of dead bodies is very rapid in a hot climate, it is important to remember that the so-called antiseptic action of arsenic is confined to the stomach and intestines, the other organs being as subject to rapid decomposition as in death from any other cause. Perforation of the stomach in arsenic poisoning is rare, but a few cases have been recorded in India.

The so-called " nervous cases " of arsenic poisoning are of importance, as they may be very misleading.

As an example may be quoted the case of a man, aged thirty, to whom a poisonous dose of arsenic was given. He suffered from giddiness, faintness, coma, and suffused conjunctivæ, but had no vomiting nor diarrhœa, and he recovered. In another case all the usual symptoms of irritant poisoning were present except purging.

The onset of symptoms in acute poisoning is generally rapid, that is, within half an hour. Bedford, an authority on Indian poisoning, gives eighteen to twenty hours as the average period which elapses before death, and states that 82 per cent. of the cases die within the first twenty-four hours. On the other hand, cases are on record in which symptoms did not appear for fourteen hours, and death in the case of a single lethal dose has been delayed as long as nine days ; and even longer intervals are recorded in European text-books. In some such cases the delay has been explained by the fulness of the stomach, by sleep, or intoxication by opium or alcohol.

In one case, however, recently recorded in India, where all such causes could be eliminated, no symptoms appeared for fourteen hours. Another remarkable case is worthy of mention where, in Bombay, a Parsee recovered after having swallowed " two masses " of arsenious oxide. He passed per rectum no less than 105 grains. His only symptoms were slight diarrhœa, drowsiness, and headache.

Arsenic is not invariably fatal even when taken in poisonous doses, for of eight consecutive cases treated at the Calcutta Medical College Hospital five recovered.

Opium

The next most important poison in Indian medical jurisprudence is opium. It is calculated that 40 per cent. of cases of poisoning in India are due to this drug.

Opium is seldom used for homicide or for robbing ; it is the drug *par excellence* for suicide. It is also not rarely used for infanticide. Owing to the frequency of the opium-eating habit, the drug may easily get into the hands of children, often with serious results.

Poisoning by opium is frequently met with in hospital practice. In 193 consecutive cases of poison treated at the Calcutta College Hospital there were no less than 165 due to opium, and of these 42 per cent. died. This high percentage of mortality, in spite of a most complete and ever-ready system of treatment, points to the fact that most of them were

cases of determined suicide, in which large doses were taken late at night, and the victims were found in an advanced stage of poisoning in the morning.

In the above 165 cases crude bazaar opium was used, except in one, where the tincture was taken. We may note in passing that the large experience of the Calcutta Medical College Hospital is not in favour of atropine as an antidote in such cases.

The symptoms of opium poisoning are well known, but it is less recognised that vomiting and diarrhœa are sometimes present ; tetanus and lockjaw symptoms have been observed in the case of children poisoned with opium, and the occurrence of such might well mislead the medical attendant.

Opium is usually swallowed, but in some parts of India suicide has been attempted by the introduction of opium into the vagina.

It is seldom possible to find out the exact quantity taken. Four grains are usually regarded as a lethal dose. On the other hand, recovery has taken place even after very large doses. A curious case has lately been published where seven grains of opium were taken along with croton oil. The symptoms were entirely those of an excessive dose of the oil, and as severe as if no opium had been taken.

Opium and its preparations are ingredients of a large number of patent and quack remedies ; hence poisoning from it often occurs accidentally. One of the most important patent preparations containing opium is chlorodyne. Owing to the amount of morphine in chlorodyne, it is generally assumed that in cases of poisoning the pupils would be contracted. As a matter of fact, however, it has been recently pointed out by Powell, the police surgeon of Bombay, that in five recent cases met with by him the pupils were found widely dilated, owing to the not inconsiderable quantity of hydrocyanic acid used in these preparations.

Datura Poisoning

Two species of datura are commonly found in India and the Malay peninsula, *viz. D. fastuosa* and *D. alba*. They are practically wild plants, growing by roadsides and in the fields all over India. *D. fastuosa* (Linn.) is a herbaceous plant, natural order Solanaceæ, about 4 to 6 feet high, with widely spreading branches and conspicuous trumpet-shaped flowers. The fruit is an oblong globular capsule, 1½ inches (4 cm.) in diameter, spinous, dehiscing into two halves containing a large number of seeds, the embryo of which has a characteristic curvation. The following distinction between datura and chillies is by J. D. Gimlette : —

" In many instances the light colour and the pungent taste of the latter would be sufficient to make a distinction, but when either is cooked and mixed with food, such as boiled rice, recognition by means of the taste alone cannot be relied upon. The resemblance is most marked in the unripe seeds, but a careful comparison of the two kinds shows many morphological points of difference. The *Datura alba* seed is almost reniform in shape, having one end smaller than the other. It has been, not altogether fancifully, compared to the human ear, but the margin is angular ; size about ¼ inch (5 millimetres) in length, rather less in width ; no marked odour ; taste slightly bitter ; surface somewhat shrivelled except on the two compressed sides ; testa tough and rough, being made up of a convoluted series of thick-walled cells, so arranged as to give a pitted appearance when seen with the lens. Datura seeds in powder may be distinguished by means of the cavernous appearance of their exosperm when seen under the microscope.

"The embryo is embedded in a white oily albumen, and curved in a manner peculiar to the genus. By cutting parallel to the flattened side the embryo may be seen by the naked eye to be curved, twisted, and recurved, resembling the head of a shepherd's crook.

"A watery decoction of the datura seeds, when placed in the eye, will cause dilatation of the pupil.

"The seed of the capsicum is kidney shaped, a little shorter and wider than that of the datura, pale yellow in colour, uniformly rough, and when seen in section as described above, the embryo is found in a fleshy albumen and curved like the figure 6. Capsicum seeds in powder may be recognised by means of the application of heat, the acrid vapours of capsicin being at once detected by heating even a small portion.

"A watery decoction, when placed in the eye, causes irritation without dilatation of the pupil. The seeds of the *Datura fastuosa* resemble those of *Datura alba*. They are about $\frac{1}{8}$ inch (4 millimetres) long, of an oblong kidney shape, flatter, and darker in colour. About eight of them weigh one grain in the dried state."

The use of datura is in a special degree an Indian method of poisoning. It is very seldom used for actual homicidal purposes, but owing to a widespread belief among the natives that it is a mere intoxicant, a fatal issue sometimes results from its liberal use.

It is usually given to produce a sufficient degree of insensibility to facilitate robbery and theft. The story told by the victims is almost always the same.

It is to the effect that a party of villagers are travelling along a road. Towards evening they are met by another party of presumed travellers. One or more of the new arrivals are dressed as Brahmins, or men of high and holy caste. They make themselves agreeable, and before dark the whole party settles down to camp out for the night. One of the robbers proposes that, as he is a Brahmin, he will do them the honour of cooking for the whole party. The compliment implied is too great to admit of any refusal, and the supposed Brahmin sets about preparing the evening meal of rice and pulses. In cooking he easily manages to add a quantity of datura seeds to the mess prepared for his victims. About half an hour after the food has been eaten the symptoms of poisoning appear and soon result in a state of stupor and coma, during which the victims are helpless and easily robbed. When they come to their senses a few hours after the robbers are far away, and with them the valuables of the deceived travellers.

This form of poisoning for robbery has, to an almost complete extent, taken the place of the strangling methods of the Indian thugs, or road robbers, of an older time. Datura poisoning is now almost altogether in the hands of professionals, and such are to be found all over India. Quite recently a gang was discovered at the Howrah railway station in Calcutta. They were headed by a native policeman, and they confined their operations to the watching and following of parties of native travellers alighting at lonely, out-of-the-way roadside stations.

The following two characteristic cases are quoted by Gimlette from his experiences in the Malay States. They show that the same method of mixing the seeds in the food is practised in Malay as in India :—

"*Case* 1. In the month of April, 1896, a Malay named Saiyed was charged at Kuala Lipis, Pahang, with causing hurt by means of poison. He pleaded not guilty, but although the motive of his crime was never actually discovered, he was eventually convicted of having mixed datura seeds in a curry, thereby stupefying a Malay constable, his wife, their niece, and a girl friend, as well as two men, all of whom happened to eat of the same dish. The symptoms in each case were similar, namely, attacks of giddiness, passing into unconsciousness for a few hours, followed by complete recovery. This group of cases is of some interest owing to the fact that one of my colleagues, who appeared for the prosecution, was able to give evidence of a very practical kind. A sample of seeds and powder which had

been found in the prisoner's handkerchief was sent to Dr. D. H. McClosky for identification. I am indebted to him for the following notes of his personal experiment. He says: 'I took pinch doses of the sample, which consisted of the bruised seeds, and had the following experience: I felt flushed, dry about the mouth and throat, and became hoarse. When I tried to walk I staggered about like a drunken man and got very excited. I then took an emetic of zinc, vomited, and slept for about five or six hours.' Dr. McClosky is further said by an eyewitness to have been in a delirious state, rolling on the floor, and uttering inarticulate cries.

"*Case* 2. *Datura Seeds Mixed in Food by Pathans.* In February, 1900, a Pathan was found by the roadside in Kuala Lipis, and brought to the hospital about midnight. He was half conscious, very restless, had dilated pupils, and was supposed to be intoxicated with alcohol. Mr. Henry Philips, the chief dresser, who saw him the next morning, fortunately suspected datura poisoning. He applied the stomach-pump, and found datura seeds, together with partially digested rice and green peas, in the gastric contents. The patient made a complete recovery, and two of his fellow-countrymen were subsequently sentenced to rigorous imprisonment for having administered the poison with intent to facilitate robbery."

The symptoms of datura poisoning are those of belladonna poisoning. When the seeds are given internally the professional thief seems to know the exact dose necessary. The symptoms of unconsciousness may commence early—within a quarter of an hour. The effect of the drug may last for two days, and is more severe, as a rule, if it is administered during childhood and old age. Many fatal cases of poisoning by the fruit and young seeds of datura are reported by Chevers, as well as five fatal cases of poisoning by the leaves. They are mostly in children and aged persons. Excessive dilatation of the pupil may be regarded as a dangerous symptom in datura poisoning. The loss of power of accommodation which is thereby produced, and the hallucinations caused, may explain the disorders of vision which are so common. There is also a certain degree of impairment of memory before complete recovery.

In India this after-effect has been regarded as one of the causes of insanity, and it seems not at all improbable that slow or chronic poisoning by datura would tend to weaken the intellect. In the absence of any suspicious circumstances, diagnosis may be difficult; cases have been confounded with rabies, delirium tremens, and mania.

The Cocaine Habit in India

It is a suggestive and somewhat remarkable fact that some years ago the practice of taking cocaine became widely prevalent in many parts of India, in fact to such an extent as to necessitate special legal measures for the control of the sale of this useful drug.

In 1913, in one province alone, no fewer than 34,366 packages of cocaine were seized by the customs and police.[1]

The drug is usually taken for its euphoric effects, mixed with *pan* and *betel* so commonly eaten as a masticatory by the natives of India. The cocaine is usually eaten in the form of the hydrochloride, as used in ophthalmic practice. The dose is generally about one grain, and is repeated as often as the *habitué* is able to buy this expensive drug. It produces a temporary feeling of satisfaction and well-being, but is soon followed by a reaction which calls for repetition of the drug. Though I have seen individuals who claimed to be in the habit of eating as much as half a drachm a day, yet I am bound to say that in not less than one hundred cases in which, on admission to prison, the drug was immediately

[1] Waddell, "Medical Jurisprudence," p. 670.

and certainly stopped, the symptoms of abstinence were slightly marked. Beyond a temporary depression and a hollow feeling in the abdomen there was little complained of. One distinguishing sign of the cocaine-eater (at least when it is eaten along with lime *pan* and *betel*) is an ebony blackness of the teeth, especially on their posterior aspects. This sign I had not seen mentioned anywhere before I first pointed it out.

The recent introduction of the cocaine habit suggests the view that if the efforts of the well-meaning opponents of what is called the " opium traffic " were successful, a new drug or narcotic would soon replace the use of opium with results at least as serious.

Other Less Common Poisons

The practice of **camphor-eating** has recently been reported as not uncommon in some native girls' schools in Calcutta. Giddiness and excitement, followed by a deep sleep, result from its excessive use.

Marking nut (vernacular *bhela*), the fruit of *Semecarpus Anacardium*. This is rarely given internally. An " ink " is made from the acrid juice which is used in marking clothes. After marking the clothes it is necessary to damp the part in lime-water to neutralise the irritant, otherwise if it touches the skin it will give rise to a painful vesicular eczema-like inflammation. It has also frequently been used to fabricate bruises on the skin.

Madar (*Calotropis gigantea*), a common weed all over India. The leaves contain a white milky juice, which is acid. The root has some traditional repute as a remedy for dysentery, instead of ipecacuanha. The juice of madar is chiefly used as an abortifacient.

Physic nuts (*Jatropha Curcas*).

The tuberous root of *Gloriosa Superba* is popularly believed to be poisonous. It is so only in large doses.

Levant nut (*Cocculus Indicus*). The shape of the nut on section is characteristic.

Lal chitra (*Plumbago Zeylanica*) has been mentioned as an abortifacient. It can also be used to produce bruises artificially.[1]

Jequirity (*Abrus precatorius*). An irritant to mucous membranes, chiefly used for cattle poisoning. The pounded seeds are made into a paste and dried on sharp points of bamboo. These poisoned points are inserted under skin of cattle. The animal dies, and the hide-dealers (*chamars*) buy the hide for a trifle.

Drs. Warden and Waddell, of Calcutta, showed that the poison of jequirity is *abrin*, which acts as a blood poison.

Warden recorded a case of poisoning by jequirity introduced beneath the skin of a man by means of a *sui*, or pointed stick above mentioned. Several similar cases of its homicidal use are on record.

Nux vomica and **strychnine.** The use of this drug for poisoning is rare, and generally accidental, sometimes suicidal. Waddell[2] suggested that the mistaking of *Nux vomica* bark (*kuchila* in the vernacular) for the bark of *Holarrhena antidysenterica kurchi* might explain some of the great mortality attributed to tetanus in Calcutta.

[1] Walsh, *Indian Med. Gaz.*, January, 1900.
[2] *Indian Med. Gaz.*, March, 1885.

White oleander (*Nerium odorum*), or "true oleander," is sometimes the cause of poisoning. Some cases are reported [1] by C. L. Bose, who has isolated three active principles: neriodorin, neriodorein, and karatin. The vernacular names of this plant are *kaner* and *karabi*. The symptoms somewhat resemble those of strychnine: twitchings, convulsions, spasms, vomiting, and unconsciousness.

Yellow or bastard oleander (*pilakaner* in the vernacular, *Cerbera thevetia*). This is commonly used for suicide and as an abortifacient. The symptoms are exhilaration followed by depression and paralysis.

The peculiar triangular shape of the nut is characteristic.

Aconite. *A. ferox* is very common in the Himalayas, especially in the Sikkhim hills and on the road to Thibet. The long (3-inch to 4-inch) conical roots are known in the bazaars as *bish* or *bikh*. A decoction of the root is mixed with other liquor, and is thus used for poisoning.

[1] Ibid., 1901.

APPENDIX

The Rhesus Factor

In considering the medico-legal applications of blood-grouping procedures, brief reference was made (Vol. I, p. 419) to the Rhesus or Rh factor. It was stated that about 85 per cent. of the population are Rh-positive while the remainder are Rh-negative, and the potential value of the Rh factor in disputed paternity cases was also indicated (Vol. I, p. 423).

It is now well recognised, however, that the so-called Rh factor is not a single antigenic entity, which is either present or absent, such as appears to be the case with the previously detected group specific factors. This was first suggested by the discovery of an anti-serum which caused agglutination of the Rh-negative bloods (and a proportion of Rh-positive bloods also). Subsequently, other anti-sera were found, capable of producing agglutination in different proportions of Rh-positive bloods, and it became apparent that the precise status of any individual, so far as his Rhesus grouping was concerned, is determined in a manner much more complex than can be explained on the basis of a single pair of allelomorphic genes.

It is now reasonably established that a complex of antigens is involved, six in number, each of which is capable of inducing antibody formation, although they are not all equally powerful in this respect. Various systems of nomenclature have been devised for these antigens, and for the Rhesus Groups which they determine. The system proposed by Fisher has the advantage of greatest clarity. The antigens (or antigen-determinants) are named Cc, Dd, and Ee, and the present conception is that a member of each pair occupies one of three closely related genes in the relevant chromosome. Eight combinations are therefore possible. These combinations, with, in brackets, the Rh designation assigned to them, are as follows :—

CDe (Rh_1)	cDE (Rh_2)	CdE (Rh_y)	CDE (Rh_z)
cDe (Rh_o)	Cde (Rh′)	cdE (Rh″)	cde (rh)

In practice, the commonest combinations are those which are underlined in this list.

Since one such chromosome is derived from each parent, the complete Rh group of an individual must be indicated thus : CDe/cde, CDe/CDe, CDe/cDE, cde/cde, etc. ; or alternatively, thus : Rh_1rh, Rh_1Rh_1, Rh_1Rh_2, rhrh, etc. A parent may pass on to his offspring one or other of the antigen combinations which he himself possesses, but it is to be noted that the combination of three antigens is transmitted as a single entity.

The antibodies corresponding to the six antigens have also been variously named, but they are most easily comprehended if referred to as Anti-C, Anti-D, Anti-d, Anti-e, etc. These antibodies do not occur

naturally in human serum. Their formation is induced by the entry into the circulation of a Rhesus antigen which is foreign to the individual concerned, either in the process of blood transfusion, or, in the case of pregnant women, from a fœtus containing such an antigen.

All six antigens must be considered capable of stimulating antibody formation, but, as already indicated, they are not all equally liable to do so. Antigen D is by far the most potent in this respect. The great majority of clinical cases of Rhesus incompatibility are due to iso-immunisation against this antigen, and all individuals possessing this antigen are "Rh-positive."

The term "Rh-negative" should only be applied to individuals of genotype cde/cde (rhrh), though even the antigens c, d, and e can give rise to specific antibodies. Indeed, of the six possible antibodies, five have already been demonstrated and used for the determination of the Rhesus genotype—Anti-d is the exception. From many points of view, therefore, the terms "Rh-positive" and "Rh-negative" can be regarded as obsolescent, although they still have their value in clinical practice.

The medico-legal importance of the Rhesus sub-groups lies in the greatly extended range of blood grouping tests which they render applicable in cases of disputed paternity. By a determination of the Rhesus genotypes of the parties concerned, the possibilities of exclusion are greatly increased, and as the various anti-sera become more generally available and as the basic hypothesis becomes more firmly established, the tests are bound to become more widely applicable and legally acceptable.

The technique of the tests is fundamentally similar to that required for other blood grouping purposes, but a number of refinements and precautions are essential. It is not practicable to discuss these in detail, nor is it possible to enter into a fuller consideration of the Rhesus factor in all its complexities. For further information, the increasingly voluminous literature must be consulted. The subject is essentially one for specialists, but all who are concerned with medical jurisprudence should recognise its implications.

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